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Southern Medical Association, Thirty-Sixth Annual Meeting, Richmond, Virginia, November 10-12

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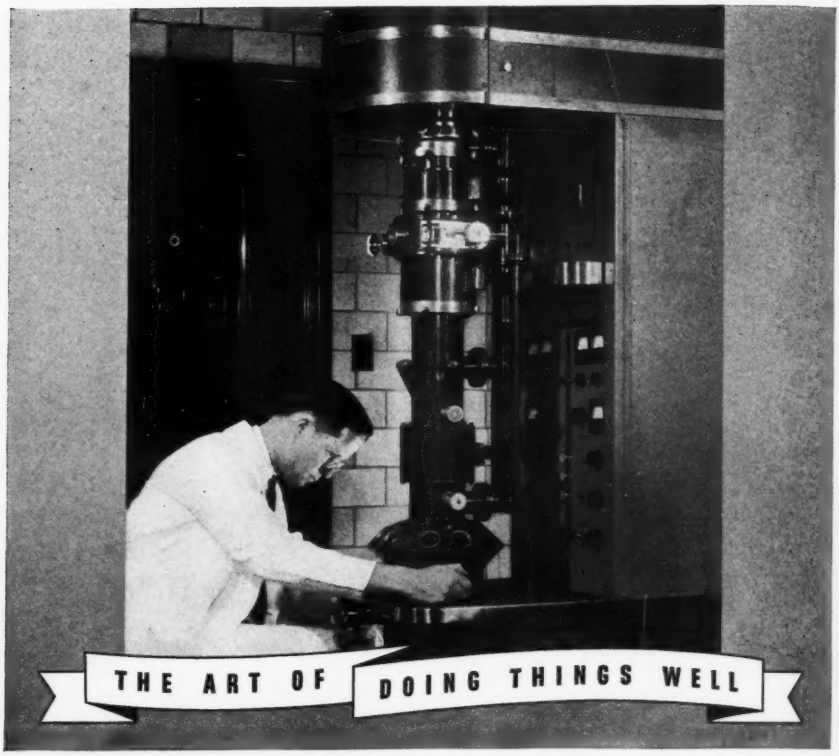

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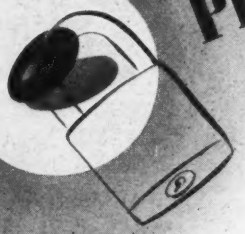

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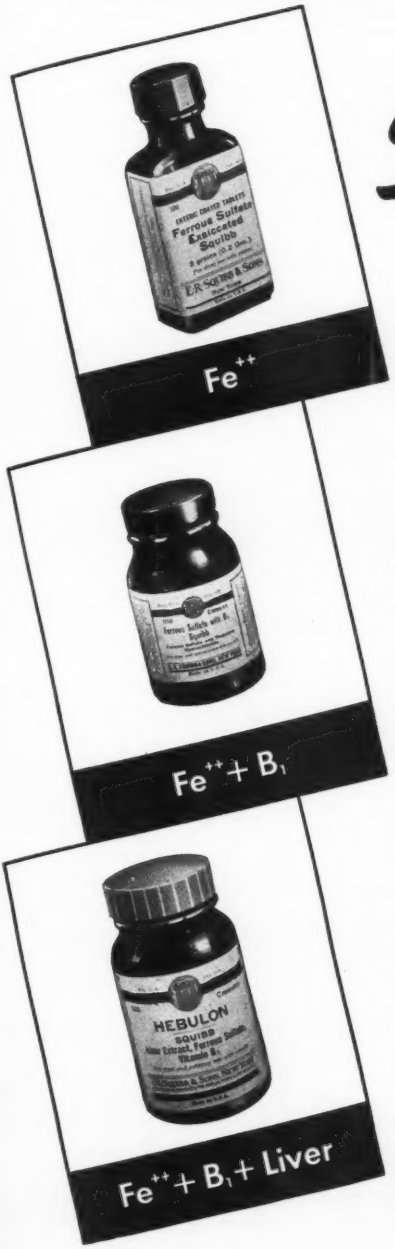

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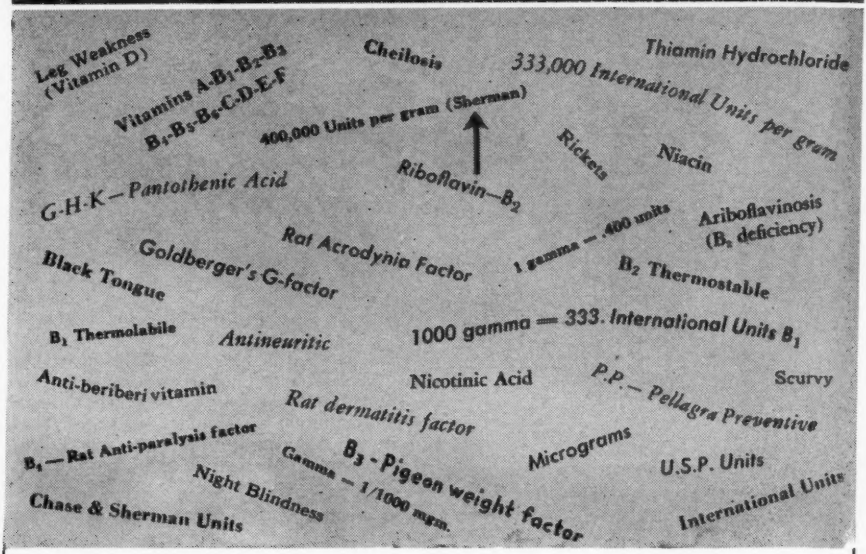

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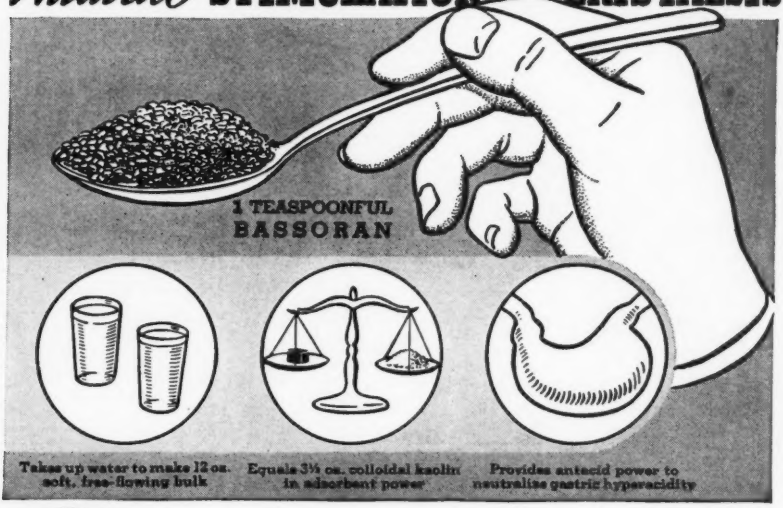
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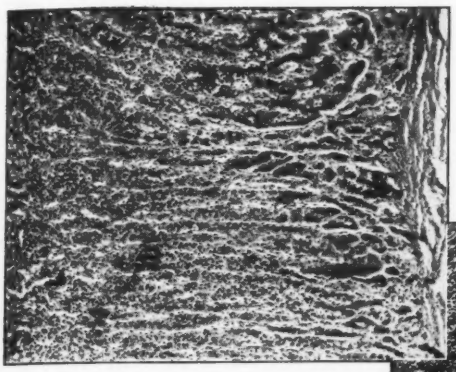
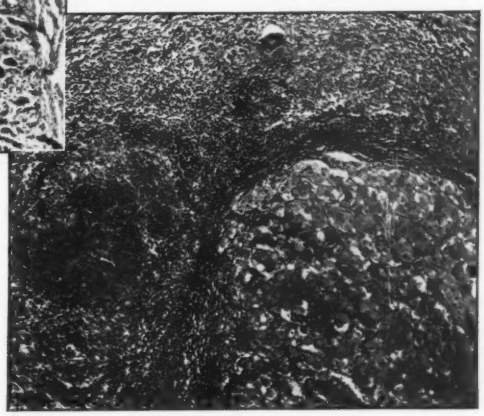


Tuberculosis of the Adrenal

The original description of Addison's disease attributed the condition to tuberculosis of the adrenal. Recent autopsy series show that there may be other causes and that these account for a considerable proportion of the cases.



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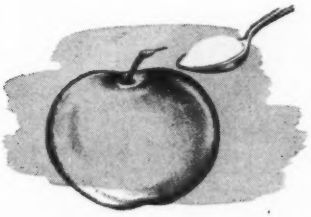

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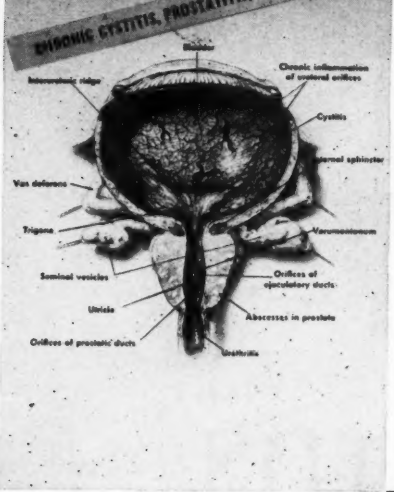

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*Clinical Application and Results of Pyridium Therapy, Illinois M.J *78:* 544-547, Dec., 1940.

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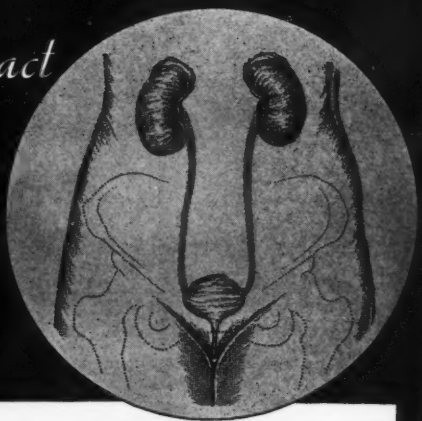

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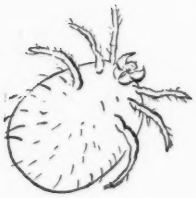

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1. ROMEO, Z. J.: Sulfur and Soap as Effective Prophylaxis Against "Chiggers" (Red Bugs) in the Army, Mil. Surgeon **90**:437 (April) 1942.
2. NOLAN, R. A.: A New Method of Treating Scabies, Mil. Surgeon **82**:52 (Jan.) 1938.

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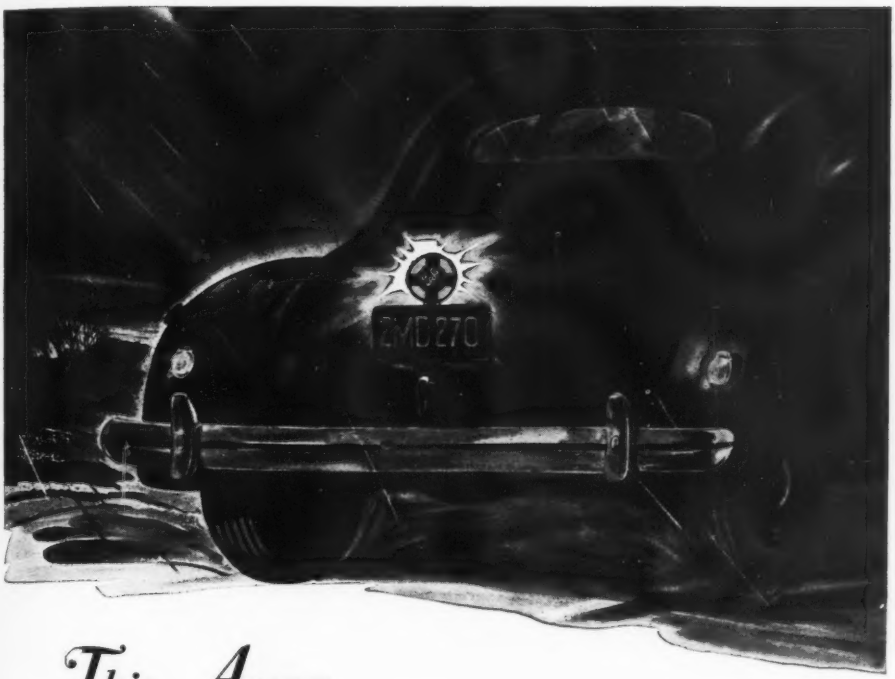

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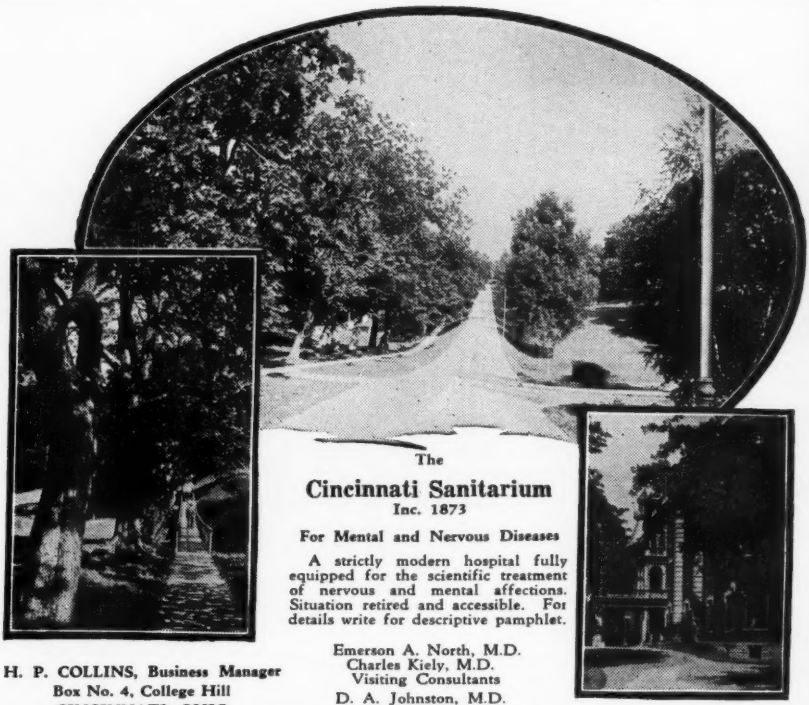

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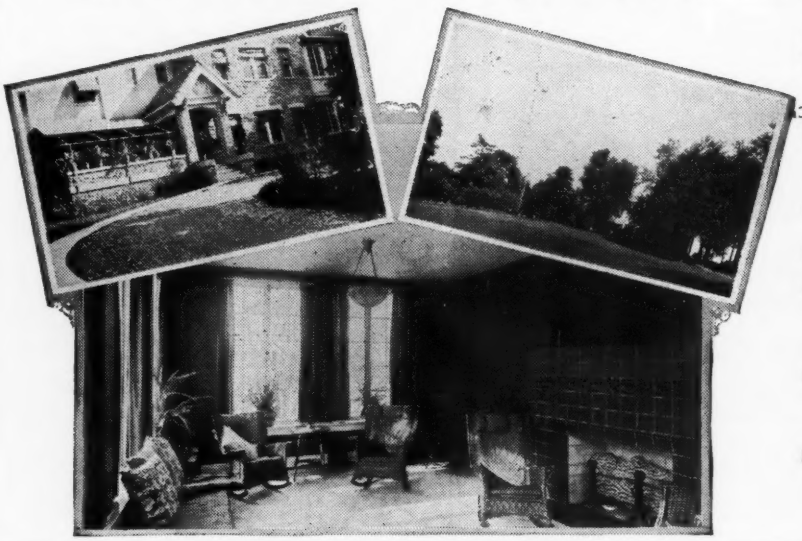

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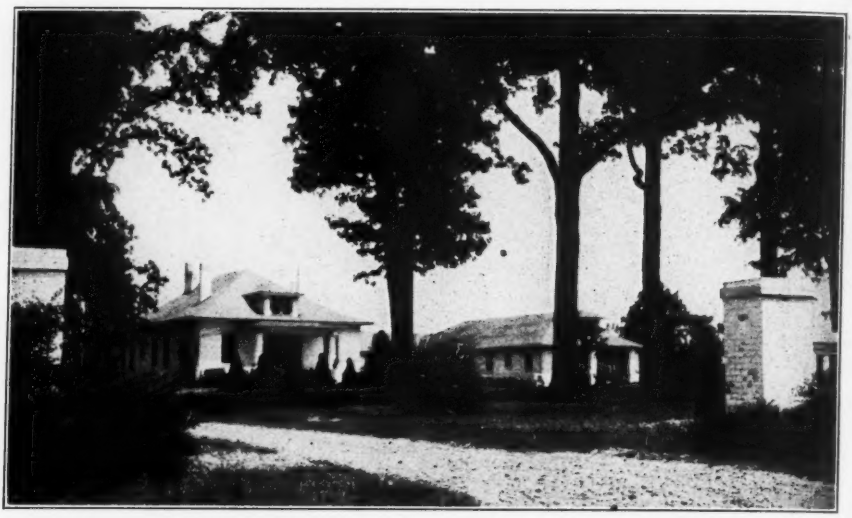

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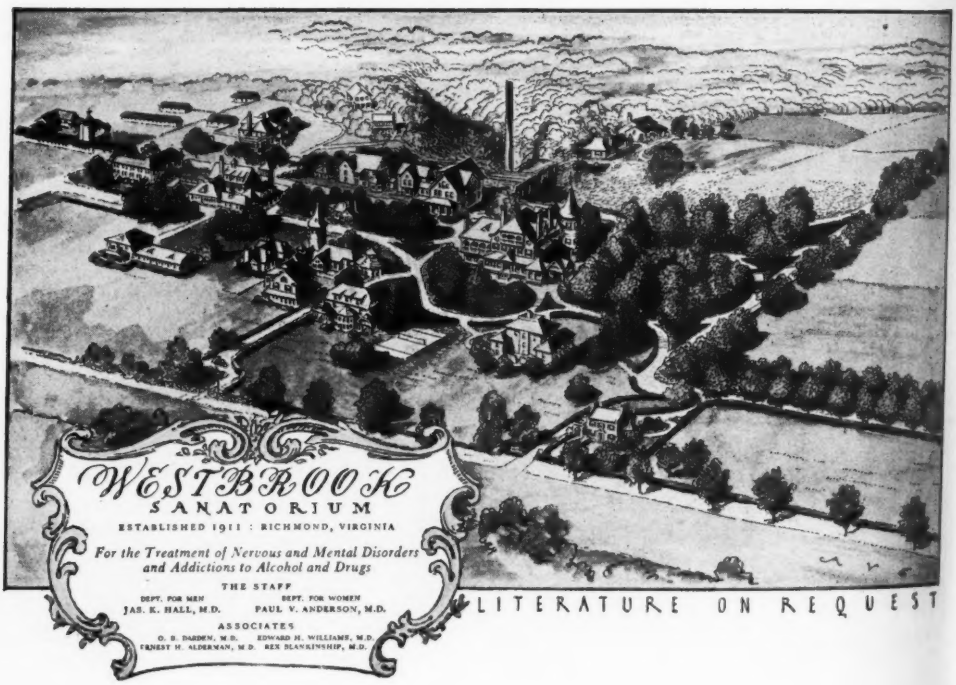
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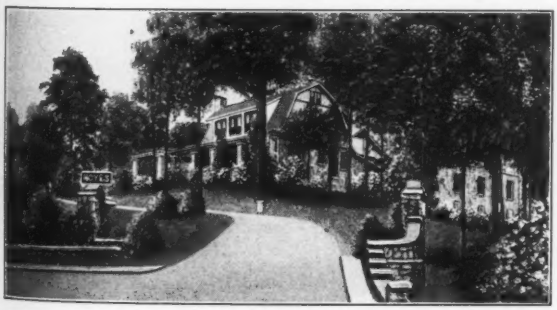

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HUMAN BITES*

AN ANALYSIS OF 90 (CHIEFLY DELAYED AND LATE) CASES FROM CHARITY HOSPITAL OF LOUISIANA AT NEW ORLEANS

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Four times within a recent two-week period the New Orleans newspapers carried items about the biting of human beings by other human beings. Three stories were strictly local. A policeman went to the aid of a fellow officer and was bitten on the forearm by the traffic violator resisting arrest. Another policeman, endeavoring to stop a free-for-all fight, was bitten on the thumb by one of the negro participants. A negro bit the nose off another negro in the course of an argument which soon became physical. The fourth item, carried by the national press associations, concerned the attempted kidnaping of a former governor of a Southern state. In the fracas the intended victim was severely bitten on the thumb by his wife, who thought she was biting his assailant. The concentration of these four items within so brief a space of time is unusual, but the appearance of such stories in the daily papers is not unusual, just as discussions of human bites as a medical entity are no longer unusual in the medical literature.†

HISTORICAL NOTE

The first human bite, in the literal sense, was reported in 1920 by Hennessy and Fletcher.[1] A thieving Malay on a rubber plantation, caught by the overseer, bit him on the forearm and left thumb, tearing off the nail and fracturing the terminal phalanx. Pilot and Meyer,[2] in 1925, and Flick,[3] in 1929, each reported the case of a negro bitten in a fight, and Fuller and Cottrell,[4] in 1929, reported a similar injury, though not in a negro. In 1930 Bower and Lang[5] reported a bite inflicted by an insane negro upon another. Severe infections followed in all these cases.

The first infection reported from human saliva was not caused by a true bite. It was reported by Hultgen[6] in 1910 and concerned a 7-year-old girl, who developed partial gangrene of the left index finger as a result of biting her nails. The following year Peters[7] reported two tooth wounds, both of which occurred in fights, when one contestant struck the other in the mouth with the clenched fist.

This was the status of human bites, or, more properly, of infections from human saliva, until November, 1930, when they ceased to be medical curiosities and became an established pathologic entity with the publication by Mason and Koch[8] of 13 personally observed cases, together with an extensive anatomic and experimental study of the routes by which infection spreads following this type of injury. It is not with the hope of adding anything of value to that important contribution, but merely with the object of putting on record a series of delayed and neglected cases that I am presenting at this time 90 cases of human tooth injuries from Charity Hospital of Louisiana at New Orleans. It is curious that except for Colby and Barr's[9] report of four cases (two in negroes) from Beaumont, and Boland's[10] report of 60 cases in negroes from Grady Hospital in Atlanta no other series seems to have been reported from the South. Almost 700 cases are now on record in the literature, and I should have expected a far larger proportion of these to have been reported from the South, where, because of the large negro population, one might expect this primitive method of injury to be quite frequent.

INCIDENCE

The majority of reported cases seem to have occurred in white subjects and in young male

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†As this paper was being completed, a New Orleans newspaper carried an Associated Press item from Milwaukee concerning the collision of two boys in a hotly contested sandlot football game. One of them was taken to the hospital with a laceration of the scalp, from which the surgeon removed one of his opponent's teeth. So far as I know, this is a type of human bite not yet reported, but worth bearing in mind, since, in addition to the risk of infection from human saliva, a certain region of the scalp is a true "dangerous area."

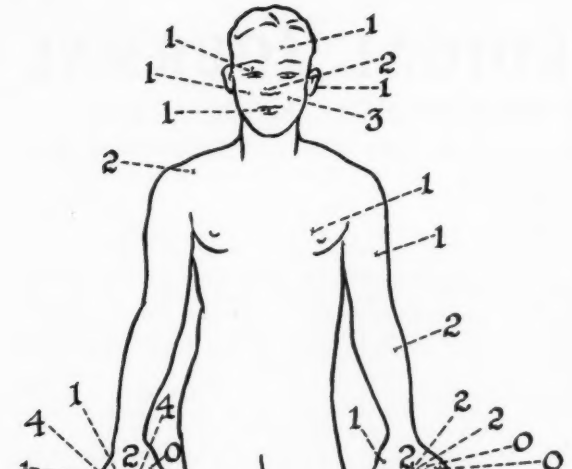

Fig. 1

Diagrammatic distribution in 90 cases of human bites from Charity Hospital of Louisiana at New Orleans. Two bites on the back are omitted from this showing; one patient was bitten in two areas.

adults, though 43 of Boland's 60 patients were women. Sixty of the 90 patients I am reporting were negroes, and 71 were males. The age range was from 9 to 68 years, and the largest number of cases (33) occurred in the third decade, though 14 patients were between 51 and 60.

There is no way of estimating the true incidence of human bites, or rather of infection following human bites, since the largest proportions of such injuries are probably not followed by complications.

Mason, discussing Boland's[10] paper, said that Koch and Allen saw approximately 75 cases yearly at the Cook County Hospital, and Bates[11] said that more than 200 bites had been observed over a seven-year period in three Philadelphia hospitals. In 1939, Barnes and Bibby,[12] reporting a fatal case, collected from the literature 342 cases, including their own, with four deaths, including their own. They seem to have overlooked the four cases reported by Colby and Barr[9] in 1932 and the 27 reported by Maier[13] from Bellevue Hospital in 1937. The total number of cases recorded to date, exclusive of recent reports in the foreign literature, which were not available to me, seems to stand at 671 cases with six deaths, the following reports having been made since Barnes and Bibby's collected series was published:

In 1939 Sniderman[14] reported an avulsive bite of the eyelids, and in the same year McMaster[15] reported 68 cases over a three-year period from the University of Southern California Hospital. In 1940 three reports were made. Fritzell[16] reported 20 cases over an approximately five-year period from the University of Minnesota Hospitals, Cohn[17] reported 35 cases with one death over an eight year period from the San Francisco County Hospital, and Boland[10] made the report already referred to of 60 cases with one death over a one-year period from the Grady Hospital in Atlanta. There has been only one report, so far as I know, in 1941; it was made by Speirs[18] from the Detroit Receiving Hospital and concerns 114 cases observed over a two-year period.

Another reason why the true incidence of infected human bites cannot be estimated is that some of the reported series include only neglected cases and some all the bites observed over stated periods of time. From personal observation I believe that tooth injuries are by no means infrequent at the New Orleans Charity Hospital, but I could not determine the true incidence there, for several reasons. The records of the accident room and outpatient dispensaries, where most early cases of this sort would be handled, are not cross-indexed, and therefore could not be used in this investigation. It was not possible, either, to locate all the cases treated in the hospital since 1923, the year in which the earliest of the 90 cases occurred. The records for some years were missing, and other records could not be found because of changes in the method of filing these injuries. The fact that 72 of the 90 cases occurred after 1930 is due, I am sure, to increased accuracy of filing and not to an increasing incidence of human bites.

SOCIAL CONSIDERATIONS

The majority of reported cases fall into two groups, those in which one individual was actually bitten by another, and those in which, as Boland puts it, the injury was received by the more civilized method of hitting one's opponent in the mouth. In the Charity Hospital series the more primitive method prevailed, 60 patients having true bites and only 25 receiving their injuries by way of blows with the clenched

fist. In the remaining five cases either the method of injury could not be determined, or it was really accidental. A 9-year-old white boy, for instance, ran head-on into another boy and his cheek was lacerated by the latter's teeth, and a colored boy, chasing a cow, stumbled and punctured his lip with his own teeth.

In the Charity Hospital series, as in other reported series, policemen were frequently bitten in the course of their duties. Smith and Manges[19] report the case of one policeman who was bitten twice within a week, by two different prisoners in two different places. Four of the 17 patients reported by Maier[13] were nurses who had been bitten by psychopathic patients. Fritzell[16] mentions a patient, not included in his reported series, who escaped from a psychopathic institution after biting off the terminal phalanges of both index fingers. Barnes and Bibby[12] state that bites are often caused by sexual practices.

True bites are most frequently inflicted in fights, particularly in negroes. White subjects are not immune to quarrels, and some are bitten in drunken brawls, but most of them receive their injuries by blows. In the Charity Hospital series a fair number of bites were received by peacemakers who intervened to settle quarrels, especially family quarrels. A negro, endeavoring to settle a fight between his sister and brother-in-law, was bitten on the left thumb by the latter, who also hit him over the head with a clock. Another negro was bitten on the arm and stabbed in the back, and still another was bitten on the forehead and stabbed three times in the chest. Colored women frequently bite each other in fights over men. A colored man reported that he was "bitten by a friend during a chat in a bar room," and another was thanklessly bitten by a sick woman he was helping to bed.

Some cases reported as bites are not bites at all. Thus Flick[20] reported an infection following the opening of a small blister on the dorsum of the hand with a knife the patient used to pick his teeth. Meleney, in a discussion of Boland's[10] paper, stated that he had seen such an infection result from plunging the hand into the trousers pocket, in which there was a loose toothpick. One of Mason and Koch's[8] patients, over a period of 18 months, maintained that his injury had been caused by a toothbrush bristle. If the story was not true, the authors point out, at least it indicated the patient's realization of the possible risk of this kind of infection.

As a matter of fact, many patients, particularly white patients in the upper social strata, are inclined to conceal the source of their injuries. Negroes, as Boland[10] notes, and as my own series shows, have no such reticences. They are perfectly willing to tell all, and if they do not, it is because the intern or resident either fails to ask them, or, more usually, fails to give them the opportunity to tell their stories.

Only a small number of cases are recorded in which nail biting, finger sucking and similar habits resulted in infection. Infection following bites by young children also is very infrequent. The youngest biter on record seems to be the 13-months-old child in Smith and Manges'[19] series, who bit his mother. Since children are often in the habit of biting adults and other children, the infrequency of recorded cases suggests that while they are in the biting stage their mouths probably contain fewer organisms than adults' mouths. Another, and probably more correct, reason is that bites by children are ordinarily in the fleshy parts of the body, in which, as I shall point out later, infection does not occur as readily as in the hand. It is theoretically possible for this special type of infection to occur in the operating room, in open wounds contaminated by droplets of saliva, but I am not aware of any recorded case.

BACTERIOLOGY

Most authorities agree that a human bite is far more serious than the bite of any animal, the difference apparently being due to the bacteriologic flora of the human mouth. Studies have shown a wide variety of organisms in the mouth, the most important being the aureus and albus varieties of staphylococci, the nonhemolytic viridans and microaerophilic varieties of streptococci, B. proteus and B. subtilis, the fusiform bacillus, and the spirochete. It was formerly believed that the infection which followed bites was caused exclusively by the fusiform bacillus of Vincent. It is now generally believed that it is due to a combined infection, in which the fusiform bacillus and the spirochete, in symbiosis, play the chief role and are responsible for the gangrene and foul discharge often observed, but in which other organisms, especially streptococci, are also concerned.

The chief criticism of all recorded series of human bites is the incompleteness or complete absence of bacteriologic studies. The Charity Hospital series falls into this category, and the few studies which were made contribute nothing of value. The most complete series, from the bacteriologic standpoint, are those of Maier,[13]

McMaster[15] and Koch and Mason.[8] Barnes and Bibby[12] studied their single case exhaustively.

There are several reasons for these bacteriologic deficiencies. Studies of the wound are frequently not made at all, and when they are, the results are frequently negative because of improper technic. Spirochetes grow poorly in cultures, though they should not be regarded as absent, as Cohn[17] warns, until both anaerobic and aerobic technics have been employed. The case reported by Hennessy and Fletcher[1] is most unusual, in that both fusiform bacilli and "enormous numbers" of spirochetes were observed in the pus in "writhing, tangled masses." The organisms were cultured successfully, but inoculations were ineffective, probably because, as the authors state, the skin was not previously damaged. Welch[21] considers the prognosis worse if spirochetes and fusiform bacilli are frequent in the smear; they are usually scanty.

In only a few instances recorded in the literature was it possible to examine the biters who inflicted the wounds, but in practically all of these cases the same organisms were found in their mouths and in their victims' wounds. The superstition familiar to all who practice in the South that "the bite of a blue-gum nigger is pizen" has no basis in fact. Boland[10] cited the studies of H. J. Harpole, who examined 32 negroes with blue gums not due to bismuth and lead poisoning, and who found Vincent's organisms in 12 and only the usual innocuous bacteria in the other 20.

PATHOLOGY

The 18 wounds in the Charity Hospital series which involved areas other than the hand (*Fig. 1*), like the similar recorded cases, presented few serious complications. This was true even when the organisms which cause serious infections were recovered. Furthermore, it is possible to treat wounds in areas other than the hand by methods which would have serious consequences if used on the hand. The reason is a purely anatomic one, and for the same anatomic reasons injuries over the knuckles are likely to be more serious than injuries caused by bites on other parts of the hand.

The anatomic and experimental studies of Mason and Koch,[8] which furnish the definitive data about this type of infection, are so extensive and so important that they should be studied in their entirety. The general background, however, is relatively simple.

The metacarpophalangeal joints have little protection but the skin, the extensor tendons, which have no definite sheaths, and the joint capsules. The relations of these various structures differ according to whether the fist is clenched or the hand is extended. When the fist is clenched, the extensor tendons are stretched over the joints, and any trauma which breaks the skin is quite likely to affect the underlying tendon and joint capsule, or the metacarpal bone itself, which knuckle is affected depending upon the angle at which the fist strikes the teeth. The extensor tendon may be injured or completely severed, the metacarpal bone may be fractured, and periosteum may be injured, or the teeth may penetrate directly into the joint.

When the hand is unclenched, the anatomic relations of the parts are immediately altered. The injured surfaces are for all practical purposes buried under overlying tissue. As a result, there comes into existence exactly the state of affairs in which the fusiform bacillus and the spirochete thrive best in symbiosis, that is, devitalization of tissue and anaerobiosis. The tendon itself has a poor blood supply, very little resistance against infection, and no power of reconstruction. Furthermore, as Mason pointed out in discussing Boland's[10] paper, the organisms of the human mouth which are deposited in the injured area are acclimated to growth in human tissues and fluids. They therefore proliferate and become invasive almost as soon as they are deposited, and are already thriving before the defenses of the host can be mustered against them.

In injuries of this sort, Mason and Koch[8] point out, the infection is likely to spread, in order of frequency, to the subcutaneous space, the subfascial space, the subaponeurotic space, the metacarpophalangeal joints, the fascial spaces of the palm, and the flexor tendon sheaths. The pathologic processes which may occur, alone or in combination, include soft tissue necrosis, abscesses, cellulitis, suppurative conditions of the tendon, lymphangitis and lymphadenitis, periostitis, osteomyelitis, septic arthritis and sinus formation, depending upon the point of invasion, the anatomic route of spread, the virulence of the infecting organism, and the time at which the patient is seen.

Injuries of the fingers, although less serious

than injuries of the dorsum of the hand, may exhibit the same type of extension and pathologic process, the differences depending upon the anatomy of the affected region. In amputating wounds and avulsive bites infection is likely to be less severe because there is free bleeding and the wound is exposed to the air, the situation thus not being the same as in deeper wounds, in which fascial planes, tendons, bones and joints are involved. Bites of the distal phalanx may result in bone felons or tendon sheath infections. McMaster reports a case in which the end of a finger, completely bitten off, was sutured in place immediately; the operation was followed by a severe felon-like infection and osteomyelitis, though recovery eventually ensued. In several of the Charity Hospital cases, as in other reported series, the distal phalanx of the finger was attached to the hand only by a few strands of soft tissue, and prompt amputation was obviously indicated.

The varieties of pathology observed in the Charity Hospital series included soft tissue necrosis, tenosynovitis, thenar and palmar space infection, and dorsal subcutaneous and subaponeurotic space infection. Bone involvement was revealed by the x-ray in 21 cases, and joint involvement in eight others. These findings are not surprising, since only three patients were seen within 24 hours of their injuries. Thirty-six were seen between 24 hours and seven days of the injury, and would be considered delayed cases, to use Welch's[21] classification, and the remainder were late cases, that is, they were seen between eight and 42 days after the injury.

CLINICAL PICTURE

One of the remarkable clinical features of human bite infection is the speed with which it occurs. In the neglected or unwisely treated case the area about the injury becomes dusky red and begins to swell within 24 hours in most cases, and in some within four or five hours after the injury. Pain is usually present, especially on motion, and is frequently very severe. Soon a thin, grayish, very foul discharge either exudes from the wound or can be expressed from it by pressure; it has the odor of pus from a lung abscess. In cases seen soon after the injury the surface wound may appear as a mere abrasion or contusion, with no teeth marks, or the bite may be clearly seen in the flesh. The appearance at a later date depends upon the time at which the patient is seen and the extent of the pathologic process, which may be strictly localized or may involve the whole hand or even the wrist and forearm also.

The constitutional reaction may be very severe, but is usually rather mild. It includes chills, temperature elevations, headache and malaise. In 32 of the Charity Hospital cases the temperature was 90° F., in 34 it was between 99 and 100° F., in 10 it was between 100 and 101° F., in eight between 101 and 102° F., and in two it reached 103° F. The pulse rate in 36 cases was between 80 and 90 per minute, in 21 between 90 and 100, in six between 100 and 110, and in five between 110 and 120.

Some degree of leukocytosis is not unusual. The blood studies in the Charity Hospital series are very incomplete, but the white cell count was above 10,000 in 30 per cent of the small number of cases thus studied, and the range was between 5,000 and 28,000. The polymorphonuclear count ranged between 67 and 98, and was over 80 in 30 per cent of the cases in which these data were available.

THERAPY

Before treatment is undertaken, it is necessary to know whether the wound is a bite or tooth injury, and if so, what is its extent. The suggestion of some authors that injuries over the knuckles should be considered as bites until they are proven not to be seems prudent, in view of Mason and Koch's[8] statement that bites are the commonest lesions in this region other than furuncles and carbuncles, which are not likely to be confused, at least in cases seen early. The possibility of bites should be borne in mind in any hand injuries, particularly those in which there is a foul discharge, and an endeavor should be made to elicit the true history. This is an extremely serious consideration. An ordinary injury should be sutured immediately, whereas closure of a human bite is the worst possible form of treatment.

Although some form of treatment had been carried out in 29 of the 87 delayed cases observed at Charity Hospital before the patients were seen there, the methods employed, chiefly compresses and other local measures, erred on the side of conservatism and were not actively harmful. This was not true in many cases in the reported series, in which suture of the wound, tight packs, and similar methods creating anae-

robic conditions frequently did a great deal of harm.

Because it is also important to know the extent and depth of the wound, some authors advocate the use of a probe. Others, of whom I am one, feel that the same amount of information can be secured with less chance of further injury by simple examination of the parts, perhaps with retraction of the overlying tissue. Speirs[18] suggests that any patient with a penetrating wound over the knuckle be asked to make a fist, so that the tendon will be brought into view and a possible injury of the joint space revealed.

As many writers have pointed out, the variety of treatments and of agents suggested for the management of human bites shows that no single method is entirely satisfactory. In a sense that is true. In another, it is not. A distinction must be made between poor results in cases seen early and unwisely treated and poor results in cases in which the outcome has been practically determined by neglect or unwise treatment before the patient is seen.

The principle of treatment in early cases is twofold: the conversion of the anaerobic state to an aerobic state in which oxygen has full access to the tissues, plus the excision of devitalized tissue in which the fusiform bacillus and spirochete might readily grow in symbiosis.

My own preference is for the thorough cleansing with soap and water suggested by Mason and Koch.[8] This is carried out for 10 minutes, after which the wound is thoroughly irrigated with physiologic salt solution or clear sterile water before any attempt is made to determine the depth of the wound by the methods suggested. Devitalized tissue is then excised under anesthesia, well beyond the area of injury, while the blood supply is cut off by a blood pressure apparatus on the arm, which should be kept in place as short a time as possible. No attempt is made to repair injured tendons or to carry out any plastic work because of the risk of sloughing. If the joint capsule has been opened, Welch's[21] suggestion is good, that collar incisions be made in the adjacent web spaces to secure adequate drainage. The injured part is splinted, in hyperextension if the extensor tendon or joint space has been injured. Compressing is carried out cautiously for the first several days, not to the point at which edema and maceration would occur, and the dorsum of the hand is directed downward to promote drainage by gravity.

If purely conservative measures have been used, it is a wise plan to keep the patient in the hospital for a period of 24 to 48 hours, beyond which time infection would not be likely to develop.

A great many antiseptic agents have been suggested for compressing and dressing the wound. It would be hard, in fact, to mention one which had not been employed in the Charity Hospital cases. Meleney,[10] who is in favor of complete débridement of the wound, regardless of when the patient is first seen, advises zinc peroxide, with which he has had remarkable success in the treatment of gangrenous wounds. Maier[13] had good results with it in several cases and discontinued its use only because he could no longer get a satisfactory preparation, but Fritzell[16] did not find it useful. Dakin's solution is inconvenient to apply by the proper technic, because of the small area of the wound, and is painful in acute cases. Sulfanilamide may be of value locally if the streptococcus is a secondary invader, but its oral use seems questionable, since it has no demonstrated effect against the fusiform bacillus or the spirochete. Neoarsphenamine, which was used intravenously in 42 of the earlier Charity Hospital cases, is now believed to be useful only as a local deodorizing agent. Fritzell[16] advises cod liver oil as a substitute for hot compresses after healing has begun. He also suggests an inquiry into the pH of human saliva, in line with the finding of Rosenthal and his associates that animal saliva contains sodium carbonate, which may inhibit motile forms of mouth organisms. It is now rather generally agreed that physiologic salt solution is probably as effective as any antiseptic agent, and Welch's[21] point is well taken, that all antiseptics are merely adjuvants to and not substitutes for surgical measures.

Various other methods of treatment have been suggested for early cases. Lowry[22] advises chemical cauterization with fuming nitric acid and Bates[11] electrocauterization. Both have had excellent results, but the methods seem theoretically open to objection as creating the very state of anaerobiosis and tissue destruction in which bacteria thrive. Smith and Manges'[19] x-ray treatment, with which they have had good results in a small number of cases, is not open to this objection, and can be used with other forms of therapy, including surgical measures.

What is done in later cases depends upon the pathology present. Simple compressing is sometimes effective, particularly if cellulitis and lymphangitis are present, but as a rule more radical measures are necessary. Sutures should be removed if they have been used, and the wound should be widely opened under anesthesia, with drainage or débridement or both carried out according to the indications. Amputation should be done without delay if the x-ray shows irreparable bone involvement. Whatever plan is used, the patient must be hospitalized and kept in bed, with the hand immobilized. Constitutional treatment is carried out as necessary. It is important that infections due to bites be treated according to the anatomic principles which would be followed in any other hand infection, and that the incisions, for the sake of future function, be as conservative as is consistent with adequate drainage.

In the Charity Hospital series the methods of treatment, alone or in combination, were those already mentioned. Thirty-six patients were treated by incision and drainage, five by débridement, two by osteotomy, three by partial amputation of a finger, and 21 by complete amputation of a finger, in four instances with partial removal of a metacarpal bone also. In one instance amputation of the arm was necessary. The period of hospitalization was only one or two days in 24 cases, but in the others was between one week and seven weeks except for one patient, who was hospitalized for 87 days.

One patient died, a 60-year-old white man who had been injured over the second metacarpophalangeal joint three weeks before. The tendons were sloughing and he had a cellulitis and lymphangitis, with a temperature of 100° F. and a pulse rate of 112 per minute. Incision and drainage were carried out at once, the index finger was amputated and the second metacarpal bone was excised on the twenty-seventh day, and further débridement was done on the thirty-eighth day. All operations were done under ethylene anesthesia. The infection was not controlled, and the patient died of pneumonia on the sixty-seventh day of hospitalization and the eighty-eighth day after injury.

Since Charity Hospital has no follow-up system, I have no late reports on these patients. The conditions present when many of them left the hospital, however, my personal observation in occasional cases, and the reported cases in the literature all make it clear that poor results are almost the rule in delayed infections from human bites. In addition to the mutilation necessary in more than a third of the Charity Hospital cases in which the hand was involved, there is no doubt that many of the remaining patients in whom more conservative therapy seemed justified had complete or partial loss of function because of scar tissue or ankylosis of the affected joints.

In the cases in which the hand is not involved, little more is needed than cleansing, compressing and suture of the wound as indicated. As has been pointed out, this type of treatment seems safe, for anatomic reasons, in avulsive and other tooth wounds which do not involve the hand.

SUMMARY AND CONCLUSIONS

(1) Infections due to human bites or injuries by human teeth constitute a definite pathologic entity, which is by no means infrequent. To the cases already on record a series of 90 cases, all but three of which were delayed or late cases, is added from the New Orleans Charity Hospital.

(2) Although bacteriologic studies in most reported series are incomplete, there is no doubt that the organisms in the human mouth, chiefly the fusiform bacilli and spirochetes in symbiosis, various streptococci, and sometimes other organisms, are responsible for the seriousness of this type of infection. For anatomic reasons bites on the hand, particularly over the knuckles, are most serious of all, because the causative organisms thrive best in the conditions created by such injuries, namely: devitalized tissue and anaerobiosis.

(3) Treatment of early cases is based upon two principles, the conversion of the anaerobic state to an aerobic state, and the excision of devitalized tissue in which bacteria might grow in symbiosis. Treatment of late cases depends upon the conditions present, and frequently must be mutilating.

(4) Human bite infections are potentially serious. They may incapacitate the patient for long periods of time, and they frequently end in functional disability or actual mutilation. Occasional fatal cases have also been recorded. These results, however, rarely occur in promptly recognized and properly treated injuries, and the remedy for this unfortunate state of affairs is therefore the treatment of early cases on the principles just outlined.

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DISCUSSION (*Abstract*)

Dr. James Barrett Brown, St. Louis, Mo.—There is little to add to this very interesting paper; it is a splendid presentation of an unromantic type of injury.

There is a question of whether or not the use of sulfanilamide might make these wounds better and easier to take care of. In infections about the mouth it is of great value. In injuries when used early it seems to have some protective influence. Whereas we used to drain fractures of the lower jaw externally we still have the rule, but we do not drain the wound if we can get enough sulfanilamide into it. The hand, of course, is bad, because there is no place for infection to go except into dangerous areas; as far as tooth punctures are concerned, even in those small areas it is possible that sulfanilamide might help. Under the mayhem law, a bite may be serious for the biter also. Once in a while a rather innocent looking person has to go to a penitentiary who has unfortunately bitten off part of an ear or nose during a state of inebriation.

Dr. Nathan A. Womack, St. Louis, Mo.—One usually notices in these infections, and all that I have seen have been on the hand, a very peculiar odor; it is an odor suggestive of pulmonary suppuration, particularly the gangrenous type.

Some ten years or so ago, Dr. Varney, of the Department of Bacteriology at Washington University, in doing bacteriologic studies on pulmonary suppuration, was able to show that in the large majority of cases there was an organism present that tended to form melanin, known as the Bacterium melaninogenicum. When this organism is grown in blood agar it gives the peculiar fetid odor described. For that reason we have made cultures of the last few human bites we have had, some half dozen or so, and in every one of them have been able to isolate this organism. It has some of the same clinical characteristics as the Vincent's organism, one being that it will grow only in symbiosis with some other organism. It has to do with necrotizing types of infection and we have never been able to recover this organism in the human bite wound unless there was a considerable amount of necrosis present.

Like the Vincent's organism, it is easily controlled if one can do away with necrosis. As a result, in the last two or three instances that I have seen in which treatment was delayed and there was no lymphangitis, but a localized area of necrosis, I have had no hesitancy in excising some of the necrotic tissue providing wide drainage and giving plenty of aeration to the wound. When that was done it seemed that it made no difference what sort of treatment we used as long as the hand was put at rest. Generally hot saline packs and splinting were sufficient.

I think the surprising thing about such wounds is the ease and rapidity with which the infections clear up once they are treated in an aggressive way. It is an infection that one cannot be too conservative in handling once necrosis is defined.

APPENDICITIS IN KENTUCKY*

By J. B. LUKINS, M.D.
Louisville, Kentucky

For a half century, the story of appendicitis has been common knowledge with the medical profession. The cause and the remedy are well known. The basic principles of diagnosis are well established. In spite of these facts, slightly more than twenty-five thousand persons die in the United States every year from appendicitis. When poliomyelitis or automobile accidents reach anything like these proportions, they make large headlines in the daily press. In several large cities and a few state medical societies, an effort has been made within the profession to do something about this alarming mortality. Intensive programs in Cincinnati, Atlanta, and Philadelphia asked the cooperation of the general prac-

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titioner, the family, the druggist, nurse and surgeon, in an endeavor to lower the mortality. Results of these campaigns showed a decided downturn in the mortality rate.

Ignorance of the public, indifference of the profession, improper management of the case, have all been stressed as main reasons leading to a fatal ending. Each of these no doubt contribute its part, but in an effort to ascertain the primary factors, we have undertaken a survey of the approved hospitals of the state of Kentucky.

At the Louisville meeting of the Southern Medical Association last November, Dr. Guy Aud[1] made an interesting report on appendicitis for the year 1939 from all the hospitals of the city of Louisville. It is interesting to note that this report compares very favorably with that of other cities.

Two years ago, the Southern Medical Association appointed a committee from each state to investigate conditions in the respective states as to appendicitis, and to make a complete report. It is impossible to say just how much progress has been made in this compilation, but a full investigation and report is yet far from completion.

Medical science is always moving, and a report of this character to be of any benefit must record the most recent data.

To receive the fullest cooperation from the hospitals, the questions asked should be simple and direct, no more than absolutely necessary to obtain the essential material. Assembling the charts, classifying the cases, and preparing a detailed report is a task requiring a great deal of time and considerable experience. We must not ask too much of hospital authorities and their assistants, if we expect their fullest cooperation.

A complete survey of appendicitis in the state of Kentucky proved quite an undertaking. This being the first survey made, it naturally is not complete, but we were surprised and delighted with the cooperation of the hospitals as a whole. A more comprehensive report to this society about every second year would be most beneficial.

The questionnaire sent to the various hospitals was short, asking only seven questions. These questions were very plain and practical, and we believe vital to the subject.

An important point to emphasize in the diagnosis is that appendicitis does not begin at McBurney's point, but the pain is usually first above the umbilicus; it does not localize at McBurney's point for from twenty-four to seventy-two hours.

APPENDICITIS INCIDENCE IN KENTUCKY

Death Rate

1920 8.2 per 100,000 population

1940 10.1 per 100,000 population

The mortality was higher last year in Kentucky than it was twenty years ago. These figures do not vary much, if any, from those reported from other states or other cities. Perhaps it is this increase that has prompted a renewed discussion of the whole subject of appendicitis

DEATHS IN LOUISVILLE, 1940

White	35
Colored	10
Total	45

This does not include deaths of nonresidents of city.

This refers to the city of Louisville alone, in which there were forty-five deaths in the year 1940, not including deaths from cases that were not residents of the city. This is in all hospitals, including the City Hospital.

QUESTIONNAIRE TO HOSPITALS

(1) Total cases, 1940
(2) Time from onset to operation
(3) Number of cases ruptured
(4) Number given purgatives
(5) Is stump routinely buried
(6) Sulfanilamide powder used in abdomen
(7) Mortality

These questions were asked of every hospital in the state.

Number of hospitals reporting	26
Total cases reported	4,081
Number of deaths	95
Mortality rate	2.3 per cent

We were very gratified at the number of hospitals that replied to our request promptly and well. There were twenty-six hospitals reporting, which includes every important hospital in the state with one or two exceptions. These hospitals by this response have proven that they are making a sincere effort to keep adequate records. It would not have been possible for them to have made so complete a report if their records had not been accurately kept. I am sure that I speak for this entire body when I express the utmost appreciation for the splendid work they are doing.

The total number of cases reported was 4,081. Only those cases in which the primary disease was appendicitis were considered. At first glance the mortality rate of 2.3 per cent seems rather high, but this is about in line with most states, and considerably lower than some.

LENGTH OF TIME FROM ONSET OF SYMPTOMS TO OPERATION

Under 48 hours 1,554 56 per cent
Over 48 hours 1,205 44 per cent

This slide, length of time from onset of symptoms to operation, really tells the story. Under forty-eight hours, there were 1,554 operated, which is 56 per cent of the whole. Over forty-eight hours there were 1,205, which is 44 per cent.

Practically all the mortality in acute appendicitis comes in those cases operated upon after the first forty-eight hours. Barring pneumonia and a few rare surgical accidents, there should be no deaths in any cases operated upon promptly.

Number of ruptured cases 476
Number given purgatives 474
This represents one out of every 8.6 cases

No claim is made that every ruptured case had had a purgative, or that every purgative given caused a rupture, but the similarity in number is so striking that it becomes almost startling. It is easy to believe that a greater number than this had had purgation, but 474 admitted it. The mortality in all ruptured cases in the city of Louisville was 16 per cent. This figure or a little higher is probably about the rate for the entire state, but we do not make this as a positive statement, as it was not possible to compute accurately the percentage rate in all the ruptured cases. A review of the literature shows that this percentage, one in every six, is almost the identical average over the entire country. There are many reports, however, from first class hospitals in other states, showing a mortality as high as 50 to 60 per cent.

IS IT CUSTOMARY TO BURY THE STUMP?

Yes 74 per cent No 26 per cent

These percentages necessarily had to be computed as to the number of hospitals in which it was customary. They do not refer to individual surgeons. Whether or not the stump was buried shows no appreciable difference in the results in these 4,081 cases.

IS SULFANILAMIDE POWDER USED IN THE ABDOMEN IN SUPPURATIVE CASES?

A new development, not generally used.

It was not expected to get a complete report on the use of sulfanilamide, since its use in this manner began only a year or so ago. In our own series of cases, no deaths have occurred since we began its use about twelve months ago. Drs. Thompson, Brabson, and Walker,[2] in an article in *Surgery, Gynecology and Obstetrics*, report 741 cases in which sulfanilamide was used intra-abdominally. They report very excellent results. To use their words,

"The convalescence and cure of these patients was so dramatic, that one could not help but be impressed with the value of the drug used thus."

They believe that the drug applied locally in this manner is absorbed at a very definite rate, and that the body absorbs it much more rapidly from the peritoneum than it does from the muscle or fat. The point of maximum absorption into the blood stream is reached in twelve hours. The amount used is 8 grams intraperitoneally and 4 grams in the abdominal wall. In cases of appendical abscess a larger amount is needed.

CHIEF CAUSES OF DEATH

(1) Delay
(2) Purgation

Purgation in all abdominal disturbances, including typhoid fever and appendicitis, was so freely taught and practiced when most doctors of my generation were in medical school that it is no small wonder that it is taking the space of one whole generation, and probably two, to learn to abstain from this pernicious habit. Preoperative purgation in some form is almost universal in children under 12 years of age. A prompt diagnosis by the family doctor will do more to prevent deaths in appendicitis than any other one factor in the whole category of remedies. A busy and successful general practitioner has made the statement that the prompt administration of a hypodermic of morphine in acute abdominal pain is usually successful in preventing a proper diagnosis in the first twenty-four hours.

The ravaging inflammatory processes, including perforation and peritonitis, are taking place under the lulling influence of an ice bag and an opiate. If all cases could be diagnosed and operated upon promptly, there should be no mortality. There is practically no mortality in the first twelve hours, and it increases rapidly after a delay of forty-eight hours.

In all cases of diagnosed rupture, the controversy between the wisdom of immediate and delayed operation still persists. In our own private work, we practice almost without exception immediate operation. By immediate is not always meant in the first hour or two, but within the first six to ten hours. Many cases have been transported long distances, they are extremely toxic, with a rapid pulse and distended abdomen. Restorative measures such as blood transfusions and intravenous glucose with sedatives will in many cases in a few hours increase perceptibly the chances of a successful operation. In all such cases cyclopropane or spinal is the anesthetic of choice.

We all know, and the reports of these hospitals show, that a ruptured appendix is practically always followed by either a circumscribed or a spreading peritonitis. The peritonitis is the cause of death. The prevention of the development of peritonitis is the big task of the surgeon. A certain percentage will be ruptured during operation, but I take it that only an experienced surgeon will undertake a difficult operation of this type, and therefore this is no argument against immediate operation.

In his engaging article of last year in the *Journal of the American Medical Association*, Dr. Gatch[3] reports 135 cases in children with perforation and gangrene with a mortality of 2.9 per cent. In none of these was the operation delayed for more than a few hours. It is our usual teaching that peritonitis kills by toxemia and intestinal obstruction. Dr. Gatch recognizes this method of progression, but after a careful study, makes the interesting observation that the changes that come in the system from peritonitis are not always the result of toxemia, but that the blood is robbed of its proteins. Then sufficient water is not absorbed from the tissues, and edema results. This is the real reason why blood transfusions are beneficial.

In my private practice, by adhering to the following routine, there has been no mortality in the past twenty-four months:

ROUTINE FOR PREVENTION OF SPREADING PERITONITIS

(1) Blood transfusions
(2) Cyclopropane or spinal anesthesia
(3) Early operation, with rare exceptions
(4) Avoidance of trauma in handling of tissue
(5) Use of sulfanilamide powder intra abdominally
(6) Avoidance of gauze drains
(7) Incomplete closure of wound
(8) Oxygen inhalations

Sequence in Fatal Cases

Pain
Purgation
Perforation
Peritonitis

This story seems so plain that it will teach its own lesson, and we hope that through the profession it will be passed on to the public.

A well-conceived preoperative plan, including the cooperation of the family physician, the patient, and the surgeon is important in every acute case. Many factors are to be considered, and all play a part in forming our judgment in each individual case. If we may decide in our own minds the state of the disease and the general condition of the patient, we may more accurately decide on the best plan of procedure, including the type of anesthetic, the point of incision, the extent of operative procedure, the question of drainage, and the general support of the patient. We believe all of these are individual questions, and should be thoroughly studied and applied in each given case.

In this discussion we are purposely avoiding the question of operative technic. Suffice it to say that while appendectomy in some cases may be a simple operation, it is never a minor operation: it is always a major surgical procedure, and the public should gain no other impression.

We have presented the results of a survey of all the cases of appendicitis in twenty-six hospitals in the state of Kentucky for the year 1940. This is admittedly not a complete or thoroughly comprehensive report, but it presents the results as seen in both private and public hospitals, patients in all walks of life, men and women, old and young, black and white, rich and poor, which makes a true picture of the effects of appendicitis on the people of Kentucky for one year. I am prompted to say that if surgeons would cease talking for a while about their own special technic of operation, type of incision, kind of ligatures, method of drainage, and other much discussed subjects, and concentrate on what we have shown to be the two most common and vital causes of death, delay and purgation, the mortality figures from appendicitis could be materially improved in a short space of time.

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DISCUSSION (*Abstract*)

Dr. Jere L. Crook, Jackson, Tenn.—I wish to emphasize the fact of the absolutely inexcusable mortality rate. The only tribunal before which any medical man or any group of men can be properly judged is at the bar of mortuary statistics. Before this tribunal, each individual and each group must be tried in his own conscience and before the world. I say, then, taking the verdict of this tribunal, for the past twenty-five years we are joint criminals. We surgeons are joint criminals with the mothers and fathers and the family physicians who are responsible for delay and who are responsible for purgation. The surgeon who knows what has been depicted here today and who fails to tell it on every possible occasion to potential victims is guilty of contributory negligence in the ultimate death of the patient.

We surgeons know it. Even the family physicians do not know it, because we are too foolishly modest and too afraid of public criticism to proclaim from the housetops the two factors responsible for this horrible mortality. I began this movement in Dallas, Texas, twenty-two years ago and made an effort at the New Orleans meeting of the American Medical Association to get that great body to adopt the reduction of appendicitis mortality as one of its major objectives.

The medical men have not been so modest, and I have only to point you to the marvelous results of tuberculosis campaigns aided by the press and by the pulpit and by the antituberculosis associations, to teach the people the real nature of this disease. I have two sons who are living today because of the prevalence of the ideas regarding the handling of tuberculosis. If the same efforts can be made, and I believe they will be eventually in my lifetime or hereafter, to do that same service on the part of the surgeons of America, we will not be confronted with this appalling situation.

Twenty years ago in Hot Springs I announced that the number of deaths from appendicitis that year was 15,000. Your essayist today has said that there were 25,000 deaths last year, this in spite of improved technic, this in spite of standardized hospitals, this in spite of almost universal knowledge on the part of every man who wields a knife of proper technic. Why should we point with pride to our achievement as surgeons and as technicians and as men who operate in A grade hospitals? Why should we point to the achievements of the American College of Surgeons and bless it abroad when we are losing 60 per cent more persons today than we lost twenty years ago? Let us put the blame where it belongs. Each individual surgeon should be blamed who does not use whatever opportunity is presented to him of telling the people and the potential victims the truth about this matter.

It is up to us to get busy and do something about it and bring the impact of eight thousand doctors on this tremendous problem and thereby bring the mortality down to reasonable figures.

Dr. Malcolm D. Thompson, Louisville, Ky.—We consider it essential in a survey to have the questionnaire as brief and as simple as possible. Furthermore, it is hoped that this and similar surveys will aid in overcoming the complacent attitude toward pain in the abdomen. Abdominal pain should always be considered of serious origin. It is always serious, and frequently dangerous. Many of the more severe cases have not the characteristic signs and symptoms, and early diagnosis is therefore difficult when most needed.

All patients with abdominal pain should have an immediate, thorough examination with acute appendicitis as a possible cause ever foremost in the examiner's mind. To delay this examination or to prescribe treatment without it will lead to serious diagnostic and therapeutic errors.

As Dr. Lukins has said, the treatment of appendicitis is never a minor procedure, and only those who have studied it in its many varied manifestations should undertake its treatment.

It will further interest you to know, I think, that much of our present treatment of acute ruptured appendicitis has been learned from Dr. Robert Elman here in St. Louis. He has simplified the classification of acute appendicitis into three types for purposes of treatment. The first is the unruptured, the treatment of which is generally accepted as immediate operation; the second is the non-toxic ruptured; and the third is the toxic ruptured. The non-toxic ruptured are best treated by operation as soon as possible after admission, and a delay of over eight hours is exceedingly dangerous.

As Dr. Lukins has told us, the toxic ruptured are best treated by rehabilitating measures to be followed by operation as soon as improvement takes place. Operation is usually possible and advisable at the end of nine to twenty-four hours of this treatment. To delay operation past this initial improvement is usually unwise and may leave serious complications.

Prior to the adoption of Dr. Elman's method, we had tried both the immediate operation and the long deferred operation in the cases of ruptured appendicitis, and we had found both of them much less satisfactory than Dr. Elman's method in the seriously ill cases.

Dr. J. Shelton Horsley, Richmond, Va.—Early diagnosis and early operation are vital.

During the last ten years and ten months, to November 1, 1941, my sons and I have operated upon 1,262 cases of appendicitis. I do not include incidental appendectomies, as removing the appendix when doing a hysterectomy, but only those cases in which the appendix was either the only or the chief offender. We have had a 0.71 per cent death rate in those cases, and not a single death was due to primary appendical peritonitis. There were two deaths from peritonitis, but in both of those cases there was involvement of the bowel and resection had to be done.

We have reduced our procedure to fairly simple terms, that every case of acute appendicitis should be operated upon as soon as the diagnosis is made; and that in practically every instance the appendix should be removed.

The postoperative treatment is very important. In peritonitis or any affection of the gastro-intestinal tract we should follow good biologic rules, and give rest. One way to give rest is to remove the irritating factor, the diseased appendix; another is to reduce function of the bowel.

Proctoclysis after appendectomy is contraindicated, because proctoclysis does not induce rest, but just the reverse. It causes distension of the colon, promotes peristalsis, and induces absorption from the right colon, which, of course, is not rest. But these cases, if septic, need water and electrolytes which we give intravenously, as 5 per cent dextrose in Ringer's solution.

We never put gauze in the abdomen or sponge out the pus where there is an abscess. That merely spreads the pus around. The pus may run over the wound and the guts. It does not look nice, but it is better than to put in snug packing to absorb the infectious material and contaminate the adjacent tissue or by sponging to force the septic material into the peritoneum. We use suction continuously, but absolutely no packing when there is pus.

Burial of the stump of the appendix, in my judgment, is quite unnecessary. When the stump is inverted a piece of necrotic tissue is imprisoned in a closed cavity along with a foreign body, the suture, and the blood supply is to some extent affected by the purse string suture. This makes an ideal condition for abscess formation as Dr. H. E. Robertson, pathologist at the Mayo Clinic, has forcibly demonstrated. To be sure, the abscess usually breaks into the lumen of the bowel and so becomes innocuous, but why tempt Providence?

Dr. John D. Hayward, St. Louis, Mo.—There are many more injuries and deaths from automobile accidents today, because many more persons are driving automobiles.

I should like to ask the essayist whether the increased death rate is due to an increase in the population, hence, more cases of appendicitis. He has said that the modern method of treatment is far superior to that used thirty years ago, when we were taught to delay operation and when purgatives were given very promiscuously. Now, if our methods of therapeutics are much better (and no doubt this is true) then why do we have such an increased mortality rate from this disease?

I am not taking issue with the essayist, because I agree with everything that has been said, in reference to modern therapeutic measures.

Dr. Lukins (closing).—I believe that the program of a survey of appendicitis, of the Southern Medical Association, if it is to succeed, should have a very simplified questionnaire. I studied the Southern Medical questionnaire very thoroughly, and my secretary estimated that it would take one employe of a hospital working on that alone thirty days to answer the questions for a hospital that had 200 cases of acute appendicitis in one year.

The same percentage of the population have appendices that had them twenty years ago. There is indeed a greater incidence of infected appendices, which may be ascribed to two causes, namely, the greater number of correct diagnoses, and the greater complexity of modern living.

The figures taken from the State Board of Health of Kentucky show that the percentage of diagnosis is greater than before.

The purpose of this paper was to show how to prevent deaths in acute appendicitis. It is first the business of the family and the family doctor to make the diagnosis, to avoid purgation, and to send the patient to a hospital; and secondly, after they reach the hands of the surgeon, it is his business to take up there the prevention of peritonitis, which is always the cause of death, and in my hands the measures that were mentioned have been very successful.

ETIOLOGIC FACTORS IN PROLAPSE OF THE UTERUS*

By JOHN W. TURNER, M.D.
Atlanta, Georgia

This paper is an attempt to discuss the etiologic factors of injury to the pelvic floor and relates only to that type of prolapse which is found in parous women as the result of injury sustained in childbearing. It might more definitely bear the title "Etiologic Factors in Injury of the Pelvic Floor," as it deals primarily with this predisposing factor of uterine prolapse.

It is generally agreed that the actual prolapse is brought about by the action of gravity and intraperitoneal pressure upon an inadequate pelvic floor. There is much difference of opinion as to whether the muscular or fascial support of the floor is of greater value. Koster[1] says that he has shown

"by histologic section on autopsy and operative material, that there was no ligamentous tissue in the base of the broad ligament around or near the uterine vessels and, extending from the uterus out to the lateral pelvic wall, as described in some texts under the name of the Mackendrodt, the cardinal, or the uteropelvic ligaments."

Colcock,[2] of the Lahey Clinic, says:

"It has been repeatedly pointed out in recent years that the essential supporting structure of the uterus is the fasciae and fibroconnective tissue which extends from the bony wall of the pelvis and is inserted about the uterus at the level of the internal os. The clinical importance of this fact cannot be too strongly emphasized for it should form the basis for all surgical treatment. The denser portions of this parametrial tissue follow the larger blood vessels and are often referred to as the cardinal ligaments. As long as this structure is intact, and its attachment to the uterus and the pelvic floor is intact, prolapse is impossible."

Masson[3] says:

"Uterine prolapse is the result of lacerations of either the anterior or posterior vaginal wall, or more frequently of both, associated with tearing and stretching of other uterine supports, especially the cardinal or Mackendrodt ligaments."

Mengert[4] has attempted a quantitative evaluation of the relative importance of the various uterine supports. He took eight female cadavers, none of which had uterine prolapse and after attaching a one kilogram weight to the cervix, the paired structures attached to the uterus were severed in varying sequences and the resulting uterine descent measured. It was found that section of the round, ovarian, infundibulo-pelvic and the upper third of the broad ligaments hardly affected

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the position of the uterus in the pelvis. The pelvic floor, although it was never incised, did not hinder experimental prolapse of the uterus, and therefore could not have contributed to uterine support in any of the eight subjects. Section of the parametrial and upper two-thirds of the paravaginal tissues allowed an average uterine descent of ten and one-half (10.5) centimeters. Marked descent of the uterus amounting to actual prolapse never occurred so long as any part of the upper two-thirds of the parametrial tissues were intact and of these two tissues the paravaginal tissue, also known as the cardinal ligaments, seemed to be slightly more important, for its division allowed an average uterine descent of six and nine-tenths (6.9) centimeters as compared with three and six-tenths (3.6) centimeters following division of the parametrial tissues.

The fact that the levator ani is three to five centimeters below the uterus when the viscus is in its normal position, that the muscle is in no sense intimately connected with the uterus, and that it can exert its supporting effect, if it has any, only after a prolapse of the first degree has occurred, throws great doubt upon its value as a supporting structure. This doubt is emphasized when we remember that the greatest hindrance to the development of prolapse is the initial resistance. Once a prolapse of the first degree has occurred, its further development cannot be prevented by yielding muscles that have passed a head with a diameter three to four times that of the uterus which presses for egress. This is conclusive evidence that the muscle cannot be considered an effective support of the uterus in its normal position. The ligaments which are its most effective support are attached to the uterus along a line which encircles the outer surface of the uterus opposite the internal os. The ligaments are five, if the pubocervical be regarded as one, and they pass outward to be inserted into the pelvic wall. Anteriorly, the pubocervical ligament extends from the posterior surface of the pubis to the cervical region of the uterus. Posteriorly,

"The uterosacral ligaments pass from their attachment to the anterior surface of the second and third segments of the sacrum downward and forward on the lateral aspects of the rectum to be attached one on each side of the uterus at the junction of the supravaginal cervix and the body, this spot corresponding internally to the position of the internal os."[5]

The cardinal ligaments which are regarded as being the most effective in support of the uterus, pass laterally from their attachment to the lateral aspect of the supravaginal cervix to be inserted into the lateral walls of the pelvis. The attachments of these five ligaments to the uterus establishes a line of fixation or "fixation circle," at the level of the internal os. This line of fixation or "fixation circle" plays an important part in the mechanism of labor and injury to its supporting structures predisposes to prolapse. The term "fixation circle" is used not to designate any anatomic structure but merely to indicate a horizontal at which the ligaments supporting the uterus are attached.

The gravid uterus may be regarded as a contractile bag; closed at the top or fundus, with an opening at the lower end, containing the fetus. It is susceptible to the action of intrinsic forces, the contraction of the uterine musculature itself, and to that of extrinsic forces, increased intra-

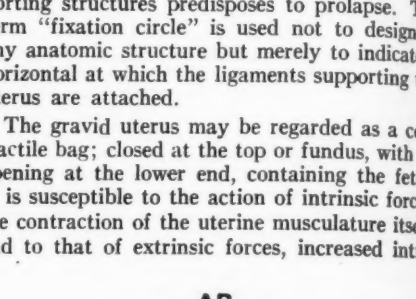

Fig. 1*
Cervix, C, and pelvic wall, P; CAP, pubo-cervical; CPP, uterosacral; and LP, cardinal ligaments.

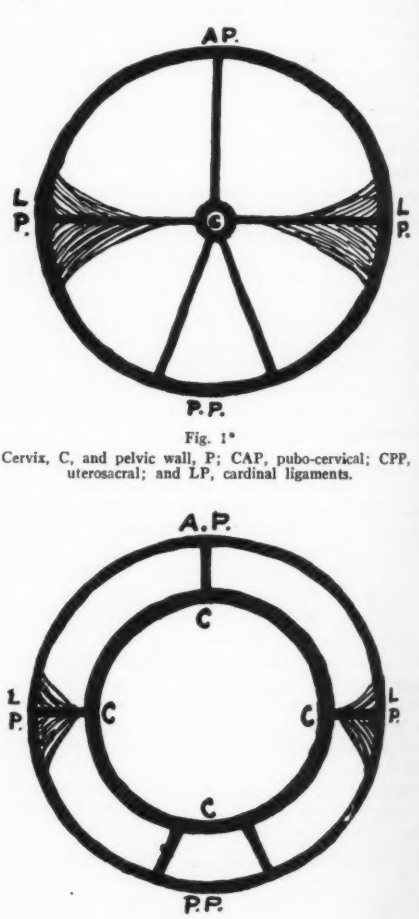

Fig. 2
The cervix, C, is now dilated and approaches the pelvic wall, P, relaxing the ligaments, which do not contract.

*I would like to express to Dr. B. T. Beasley my appreciation of his permission to use the drawing shown in *Fig. 1*.

abdominal pressure exerted by contraction of the abdominal muscles with a fixed low diaphragm, by pressure upon the fundus with the hand and by the use of forceps. In normal process of labor the forces exerted during the first stage are intrinsic forces and they are directed primarily toward dilatation of the cervix. As this is accomplished the long axis of the uterus is shortened, the external os being pulled slightly higher and the fundus descending, each approaching nearer a horizontal plane through the "fixation circle" which remains at the same relative level in the bony pelvis but ascends in relation to the baby. The statement, intrinsic forces which expend themselves against other forces within a mass or body will not effect a change of the position in space of the center of action of those forces which lies within the mass, may be accepted as a maxim of physics which is applicable to the action of the intrinsic forces of the uterus. These forces do not tend to change materially the position of the uterus and do not subject the ligamentous supports of the uterus to stress. This decrease in the long axis of the uterus, occurring predominantly above the "fixation circle," forces the baby downward and the head through and below the "fixation circle" as the external os is dilated and pulled upward. The predominant change in level takes place at the fundus, the ascent in the level of the external os being much less in extent, but definitely appreciable.

Thus the first stage of labor normally takes place without the exertion of any of the extrinsic forces and consists in the dilatation of the cervix and the shortening of the long axis of the uterus, while a point at about the level of the internal os remains fixed in its relative level in the pelvis. During this stage of a normal labor no strain has been placed upon the uterine supports and no stretching or tearing of the supporting structures or ligaments will take place regardless of the size of the baby. Also, during this stage of labor it is unnatural for the patient to attempt conscious expulsive efforts by use of the abdominal muscles and she will not do so unless urged to do so by the doctor, nurse, or some other attendant. This should never be done because an external force exerted upon the uterus at this time not only can do no good, but is capable of causing much damage. Such a force cannot aid in expelling the baby from the uterus until the os is sufficiently dilated, but as the head impinges upon the margins of the too small os, forces the entire uterus

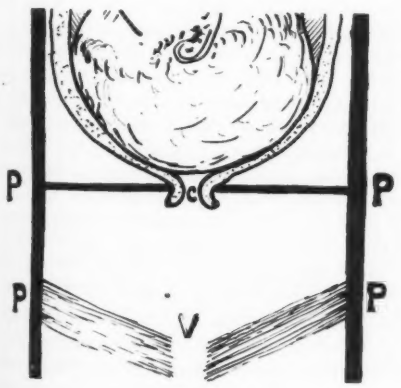

Fig. 3

Showing the undilated cervix supported by the ligaments, CP, and below, the levator ani, PV, running from the pelvic wall around the vagina, V.

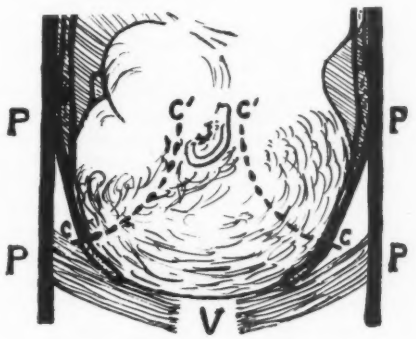

Fig. 4

The dilated cervix is supported by the ligaments, CP, and the distance, CP, is the same as in Fig. 3, but the fixation circle is lower in the pelvis. The levator ani, PV, is now supporting the uterus and head.

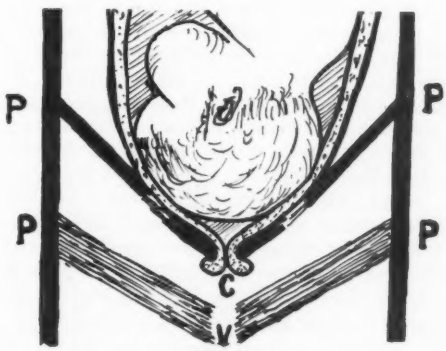

Fig. 5

Contracted cervix and small baby subjected to extrinsic forces. The distance, CP, is greater than in Fig. 3. The ligaments, CP, have been torn before the supporting levator ani is reached.

downward, carrying the "fixation circle" downward, stretching or tearing the uterine supports. Under conditions of voluntary efforts at expulsion in the first stage of labor a relatively small baby may more readily cause a stretching or tearing of the uterine supports than a large baby. The large head is delayed in its descent by impinging upon the bony pelvis and, while it is molding, allows the cervix time to dilate. The extrinsic force exerted by the abdominal muscles is transmitted by the baby's head to the walls of the bony pelvis and the "fixation circle" is relieved of this force. A small baby with a head which easily enters the bony pelvis permits a displacement downward of the entire uterus as the voluntary efforts at expulsion are transmitted by the baby's head to the still contracted cervix and must be borne at the "fixation circle."

The dilatation of the cervix which has taken place during the first stage of labor has served to afford some relaxation of the fascial supports of the uterus. The cervix may be regarded as an expansible ring placed within the circumference of another ring, the pelvis, which is fixed and not susceptible to any change in size; and attached to this second ring by a number of guy ropes or ligaments, the principle ones being the cardinal ligaments. The ligaments, like other ligaments, do not possess the ability to contract and permit only a minor degree of stretching. Upon dilatation of the central ring, its circumference approaches nearer the circumference of the outer fixed ring and the guy ropes or ligaments are relaxed, allowing the "fixation circle" to descend in the pelvis without putting any strain or tension upon the ligaments. As the cervical canal becomes fully dilated its walls approach closely to the bony pelvis and there has been sufficient relaxation of the ligamentous supports to allow the "fixation circle" to descend about four to five centimeters below the level occupied before labor began. This is sufficient to permit the head to enter the funnel shaped levator ani muscle and begin to exert an expanding or stretching pressure upon that muscle. Conditions are now different; as the fibers of this muscle, running antero-posteriorly entirely across the pelvis and being attached at its ends and not in the center to an expanding circle, are not relaxed by the passage of the head but are put upon a stretch under great tension and pressure. This pressure upon the levator ani muscle stimulates in the patient conscious efforts at expulsion and it is now that the patient voluntarily begins to exert intra-abdominal pressure. As the head reaches the pubococcygeus fibers the stimulus is more compelling and it becomes as difficult to prevent the patient from making expulsive efforts as it was to induce her to make such efforts during the first stage. It is almost as though the pressure upon the levator ani says to the patient, "Your cervix is now fully dilated and you can do no harm by pushing your baby out. Get to work." The effect of this pressure upon the levator ani muscle may be definitely exhibited by making pressure with the fingers upon the pubococcygeus fibers between uterine contractions in the patient who is sufficiently under the influence of the amnesic drugs to pay no attention to any verbal suggestions. Such pressure will cause the patient to contract her abdominal muscles just as she does when the head of the child makes the pressure.

These two factors, namely: dilatation of the cervix and descent of the "fixation circle," have been given as taking place separately in order that emphasis might be placed upon the "fixation circle." Actually, they proceed *pari passu*, the "fixation circle" descending into the pelvic canal as the dilatation of the cervix relaxes the ligaments attached to the "fixation circle." By this method the ligaments are protected from strain during the descent of the baby, the point of their attachment to the cervix ascribing an arc whose radius is represented by a line equal in length to the length of the ligament and whose center is at the point where the ligament is attached to the pelvic wall. Working in a reverse manner, as the cervix contracts after passage of the baby, the "fixation circle" is brought back to its former level in the pelvis, the point of fixation traveling in a reverse direction the arc ascribed in the process of dilatation of the cervix and descent of the "fixation circle." Even if these ligaments were rigid and attached to a flexible central ring, their ends not only could move through the same arc but would be forced to do so with dilatation of the cervix in order to maintain their ends at an equidistance from their point of attachment to the pelvic wall. Thus it is shown that the contraction of the cervix and "fixation circle" pulls the uterus back into its normal position; for, being attached to ligaments which do not stretch, it can only contract as it lifts itself back to a position which will permit the ends of the ligaments to approach the center.

Thus it is demonstrated that injury to the supporting structures of the uterus is caused primarily by the action of those forces which tend to depress the "fixation circle" and which are extrinsic forces. A large baby does not cause injury to the supporting ligaments except by

slowing the process of labor and thereby encouraging the attendant to use forceps, to exhort the patient to exert efforts at expulsion, or to resort to some other equally pernicious procedure. A large head may to some extent, by impinging upon the pelvic brim protect the "fixation circle" from the full stress of the extrinsic forces and thus prevent that stretching or tearing of the supporting ligaments which predisposes to prolapse.

This interpretation of the mechanism of labor enables us to understand why it is unnecessary for the uterus to be anchored in position by strong bands. It explains why nature resists all attempts at expulsive efforts on behalf of extrinsic forces during the first stage of labor and the mechanism by which these forces are put to work during the second stage.

It enables us to understand how the "fixation circle" can descend for four to five centimeters without subjecting the supporting ligaments to disabling stretching or tearing. It explains how the uterus is carried back to its normal position in the pelvis after labor. The same principle of muscular action that is manifest elsewhere in the body is effective here. In this instance the muscle involved is a circular or sphincteric one and its ligaments are attached circumferentially to run to a fixed insertion from which traction can be effective. It explains the supporting role played by the levator ani muscle, against whose upper surface the head of the child is being forced at the time of full dilatation of the cervix and of full normal descent of the "fixation circle." Having until this time been inactive in the process of labor, this strong and powerful muscle now has to bear the full duty of supporting the uterus while active efforts at expulsion are being made.

CONCLUSIONS

(1) The support of the uterus in the normal position is entirely ligamentous. The levator ani to which has been ascribed a supporting role lies two and one-half to four centimeters below the uterus.

(2) Tear of the supports of the uterus is dependent upon the action of extrinsic forces upon the uterus before complete dilatation of the cervix rather than upon the size of the baby.

(3) A small baby may be a contributing or predisposing factor to tearing of the uterine supports.

(4) The "fixation circle" may descend 2.5 to 4 cm. without putting tension on the ligamentous supports of the uterus, when the cervix is fully dilated.

(5) The uterus is returned to its normal level in the pelvis by virtue of the contractile muscular action of the cervix.

(6) Amnesic and analgesic drugs by preventing voluntary expulsive efforts during the first stage of labor will decrease the incidence of prolapse of the uterus.

(7) The mechanism described must of necessity be the true mode of behavior as physical laws would enforce it.

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DISCUSSION (*Abstract*)

Dr. William H. Vogt, St. Louis, Mo.—Dr. Turner has given us a very interesting, but to me not a completely convincing description of the mechanism of the production of prolapse of the uterus. Most of us believe that injury during labor to the pelvic structures, particularly to the cardinal ligaments and the pelvic fascia, is responsible for most cases of prolapse. I cannot believe, however, that his so-called fixation ring is a stationary and immovable part, and that the internal os remains fixed in its relative level in the pelvis either during labor, in pregnancy, or even in the non-pregnant woman. We all know of the important method that we make use of in testing the mobility of the cervix in carcinoma of this organ. The cervix in most normal, parous women can be brought down to the introitus or almost to the introitus, and we speak of that as a movable cervix or we say that the mobility of the cervix is limited in a given case. Nor am I of the opinion that all prolapse cases are due to long or difficult, ill-managed labors or difficult instrumental deliveries. We too often see women who have had easy labors, labors that ran along perfectly normally, in whom still a prolapse of greater or lesser degree has followed. One might argue that just because one cannot in certain instances demonstrate injury to the pelvic structures, injury may have occurred nevertheless. The explanation probably lies in weak structures in poorly developed individuals.

I am glad to hear the essayist stress the dangers associated with permitting or encouraging the patient to use her voluntary forces during the first stage of labor. This is a bad practice and, as pointed out, can do no good, but is capable of causing much damage.

Dr. Turner presents a very interesting theory of the initiation of prolapsus in the first stages of labor if the voluntary forces press the head upon the margins of the not fully dilated cervix, and he says that this is prone to force the entire uterus downward, carrying with it the fixation circle and stretching and tearing the uterine supports. He asserts that this stretching occurs

more readily when the baby is relatively small than when the child is large. Further study by others may prove his observation correct. It seems to me, however, that this likelihood would exist only if the bags of water were prematurely ruptured, and if this is the case, that might be another argument in favor of trying to preserve the membranes and not ignore them, as is frequently done today when the membranes are ruptured indiscriminately for the induction of labor. I am still one of those old-fashioned fellows who believes that the membranes were put there by nature for other purposes besides enveloping the baby in a closed sac.

By inference at least, if I understand his paper correctly, Dr. Turner seems to think that the pelvic floor contributes little to the support of the uterus and, therefore, plays a small part in preventing or producing prolapse; and yet we are all in the habit of building up the structures of this floor when we operate for prolapse.

The upper pelvic floor is a broad structure, made up of muscle and fascia extending from the bony pelvis to the uterus, and it constitutes the sustaining force of the uterus, just as the lower pelvic floor provides the supporting force. Now if the muscle and fascia of this floor are torn or overstretched and if the uterus is too heavy, as the result of subinvolution, for the sustaining structures, then the first step in prolapse, namely, retrodisplacement of the uterus, occurs. The intra-abdominal force upon the displaced uterus then pushes the uterus down further in the pelvis, and only if a good supporting pelvic floor is present will a prolapse either fail to appear or be greatly delayed in its occurrence.

Dr. John L. McKelvey, Minneapolis, Minn.—My experience with problems of birth trauma has included an opportunity to observe the Caucasian in this country, the negro in this country, the Caucasian in Europe, and the Oriental in China. You are familiar with the fact, of course, that the great site of prolapse, so far as we know in this world, is in those women in the region of Manchester in the midlands of England. Various causes have been ascribed for that. The fact that these women work in factories has always seemed to be taken as an inclusive bit of evidence. As a matter of fact, of course, women work all over the world, and in the State of Minnesota, from which I come, the women in the farming districts in particular are doing heavy work. The relationship of such work to prolapse is not at all clear.

We are seeing a good deal of it, and I have personally not been satisfied with the procedures which have been undertaken to handle the condition therapeutically. The Chinese work perhaps harder than any of this group of women, and yet in considerable experience in China we saw very little birth trauma and for practical purposes no prolapse. Something then is wrong with such a simple explanation as the etiologic relation of heavy physical work.

Now if we study the prolapses which are turning up in our groups here, we very quickly come to the conclusion that there is no continuous factor which can be demonstrated that runs through the whole of this group of cases. It is not then a simple matter of birth trauma or of physical work. We have had our attention drawn to the neurogenic factors in particular, which may be related to this condition in terms of muscle tone of the fibro-musculature, of the ligaments concerned. They are not ligaments. They are a fibro-musculature, which accounts, of course, for their tone. In view of an interest in the possible neurogenic factors, we have been very carefully taking obstetrical histories of these patients, in order at least to recognize those in whom there is no apparent birth trauma factor, for the purpose of completing the study to try to pick up a factor which may be responsible.

Let me cite a case to you. A patient who had had two normal sized babies, completely spontaneously, with normal durations of labor, came with a second degree prolapse, the uterus being completely outside. It so happened that she was divorcing her husband and intended to marry again and she was anxious to keep her uterine function. It was interesting that the patient had a club foot, which put us at once onto the story, and x-ray of the lower spine showed a spina bifida.

The patient was urged then to allow a fixation procedure to be carried out because, obviously, here was a neurogenic factor probably responsible for the prolapse. Under local anesthesia a completely adequate repair was done and within six weeks of that time, after primary healing, the patient was back again with the uterus completely outside. She was now prepared to accept sterilization and ventral fixation of the uterus, which was done without any further repair from below. After about a year and a half the support is still satisfactory.

The lack of any demonstrable continuous etiologic agent, particularly in so far as birth trauma is concerned, has perhaps unduly influenced us. The fact that we do every now and again run into a full-blown neurogenic factor has induced us to use as a basic procedure not the Manchester operation alone, but some procedure designed to hold the uterus in anteposition and to support it permanently. While it is not a routine procedure, we have come to the conclusion that a patient who can be sterilized, who has finished her childbearing career, is probably better, in the absence of a clear-cut relationship of some factor to her prolapse, with the uterus fixed to the abdominal wall in addition to the repair from below, with amputation of the cervix if necessary. A more certain support for the uterus can be so obtained.

Dr. Reis will, I know, express opposition to this view. He says that he has gone through this procedure of fixation and has moved away from it. So have we, but we have moved back.

I think then one must say at the present time that we have no really clear-cut idea as to what the etiologic agent in prolapse of the uterus is. We have subjected thirty-odd of these patients to pelvic x-ray and have been astounded by the fact that not only are the pelves not contracted in women who have uterine prolapse but, indeed, they are larger than the average pelvis. It so happens that we picked up the largest pelvis that had ever been x-rayed in the clinic among this particular group.

Dr. Turner's paper, then, is of interest. I am not sure at all that we have exhausted possibilities for studying the lesion. I am convinced, however, that any simple mechanical explanation of prolapse of the uterus leaves too much to be desired.

Dr. Ralph A. Reis, Chicago, Ill.—We should congratulate Dr. Turner primarily for a very intriguing thesis, and most of all for giving us the phrase the

"fixation circle." Those of us who have been interested in prolapse have groped for many years for some phrase that would describe that encirclement of the lower uterine segment in this transverse fascial plane, and I think the term "fixation circle" fills that need for us.

I am not quite convinced yet that it is as simple mechanically as he has made it. There are a number of other factors. We must not forget prolapse in the nullipara and prolapse in the virgin. If you take the trouble to x-ray these patients you find almost universally the neurogenic factor which Dr. McKelvey mentioned, namely, the occult spina bifida.

We have followed the problem of what to do with prolapse for a long time. In 1928, my senior associate, Dr. Baer, and I described our results in 212 consecutive patients with prolapse and we were sure we had the answer. Then we reviewed the next 212 in 1936, and we were sure we did not have the answer.

Dr. McKelvey said he was not unduly influenced by one case, but he went back to fixation because one of his Manchesters failed. I could bore you with the recital of fixations that have failed, that have reprolapsed through the vaginal tube and have dragged the parietal peritoneum and the points of fixation with them. I do not believe that a single failure, or multiple failures, in any one method should remove that method from our armamentarium. There is no one procedure that we can say today is the method of treating uterine prolapse. Uterine prolapse must be individualized. In the nullipara, the woman with no cystocele and no rectocele, the cure can be brought about much more simply by the abdominal approach. In a patient who has a large cystocele, a large rectocele, a relaxed perineum and the prolapse, which is the usual picture, the approach is much better by the vaginal route.

In describing his groups in which prolapse had occurred, Dr. McKelvey brought up the question of work. We in Chicago at Michael Reese find our greatest incidence of prolapse in Jewish women, who almost universally develop cystoceles and rectoceles after multiple deliveries and they notoriously are not the women who work. If any group of women are spoiled and sit around and play lady it is that group.

I do not believe work to be a factor. The incidence of prolapse in the Jewish half of our clientele is much larger than it is in the other group. So again there must be other unknown factors.

I should like to throw out to you for consideration the idea that Sturmdorf advanced about 1932 or 1933, in which he came closest, I think, to giving us a plausible explanation. It had to do with pelvic inclination, the degree of pelvic inclination which was present in the individual woman, and its resulting tendency to throw the uterus either forward or backward.

Dr. Turner (closing).—My treatise is limited entirely to a consideration of those cases of prolapse which occur in parous women and does not take into consideration the virginal type of prolapse. I realize further that I am indulging in speculative gynecology in giving this paper.

Dr. McKelvey referred to the fact that he found in x-raying those patients who suffered from prolapse that their pelves were larger than the average pelvis. This would show a disproportion between the size of the baby and the pelvis, the baby being relatively smaller than would be normal, unless these patients also had much larger babies than normal patients. One of these patients in whom prolapse occurred, of whose pelvis he had taken x-ray pictures, had the largest pelvis that has ever been x-rayed. There must have been quite a disproportion in size, so that the baby could have been pushed out easily; the pelvic brim offering no resistance, the force of expulsive efforts being transmitted directly to the uterine supports.

Dr. Vogt referred to the motility of the uterus, an attribute of the uterus which we all recognize and which I did not attempt to discuss because the fixation, as I referred to it is that which occurs beyond the limitations of normal motility. For several years I have asked two questions of patients coming to me suffering from prolapse: first, what was the size of your baby? Second, did you bear down, or did the attendant ask you to bear down early in labor? About 40 per cent of these babies have been less than the average in weight, in spite of the mother's tendency to exaggerate the baby's birth weight. The others have been greater than the average. To the second question they have invariably replied, "Yes, I did have to bear down. The nurse told me to push the baby out." I think you will find that voluntary efforts at expulsion have been made by every patient who suffers from prolapse as the result of child bearing.

We cannot discuss prolapse and make it a cut-and-dried subject and finish it with the presentation of any particular factor. My paper was an endeavor to put before you that factor which I believe is instrumental in the causation of the greatest number of prolapses.

ANISEIKONIA*

By LAWRENCE T. POST, M.D., F.A.C.S.
St. Louis, Missouri

The existence of aniseikonia, or unequal-sized images in the two eyes, has been known for many years. Its first description of which I am aware was by Donders in his textbook of 1863. Nothing was done about the condition, however, because no method of handling it was known at that time. Occasionally through the years a reference to it was to be found in the literature. In about 1924, Drs. Ames, Gliddon, and Ogle in Dartmouth became interested in the subject and were able to devise an instrument by which it was possible to measure aniseikonia. They have worked on this subject continually since that time. A few instruments became available about ten years ago. One of these was placed at Washington University eight years ago† and has been in use since that time.

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*Read in General Clinical Session, Southern Medical Association, Thirty-Fifth Annual Meeting, St. Louis, Missouri, November 10-13, 1941.

*From the Department of Ophthalmology, Washington University and the Oscar Johnson Institute.

†Through the courtesy of the Aloe Optical Company.

About 800 examinations have been made in the Department of Ophthalmology at Washington University during the eight years. The American Optical Company has been able to manufacture lenses, which can be ground with the usual correction of refractive errors, to equalize the unequal images and these glasses have been dispensed on prescription from the examiner. Questionnaires were sent out three years ago to the patients for whom aniseikonic correction had been ordered. The questionnaire read as follows:

Question 1. Are you still wearing your eikonic correction for the use for which it was prescribed? If not, how long did you wear it?

Question 2. Have the results been entirely satisfactory?

Question 3. If not entirely satisfactory have they been moderately satisfactory?

Question 4. Have they been unsatisfactory?

One hundred and thirty replies were received to this questionnaire. Approximately 33 per cent had received entire satisfaction. A second 33 per cent had been partly satisfied, and a third had found no benefit from the glasses. How may we interpret these results? When one considers that the patients referred to our clinic have been those who have never been able to obtain satisfaction from any glasses anywhere, in spite of the fact that many of them had their eyes examined by competent ophthalmologists, we must think that the answers reflect some real merit in the test since at least 66 per cent have benefited. In most of these patients no, or only a small change, of the essential refraction was made. To cite individual cases would take too much time and would, of course, be subject to the criticism of selection. Many of the cases were in patients who have been under our observation for many years; persons whom we have known well and among whom are several members of the Washington University staff. Their stories are too enthusiastic to be discounted entirely on the basis of psychology. It is well known that neurotic patients are often helped temporarily by almost anything that is done for them in the way of changing refraction, but most of these eikonic patients are benefited not only temporarily but throughout the years. We have tried at times to test the patients by making changes in the glasses of which the patient was not aware, but invariably he noted the difference and had to have the proper glass in order to get a return of his comfort. It has been found that very small differences in image size do not produce symptoms, nor do very large differences, probably because of tolerance in the former and suppression in the latter case.

Among the criticisms that have been made of aniseikonia, one is that aniseikonia is not actually being measured but only a retinal slip, a lateral phoria or a phoric slip, but recent experiments have demonstrated the fallacy of this. A minor criticism is that the instrument used by most technicians does not measure the difference of image size in the oblique meridians. This is true, since only an instrument which is at Dartmouth can be utilized for this purpose, but it is possible to grind glasses that correct oblique meridinal aniseikonia. In other words, we are in the same position in aniseikonia that we should be in, in simple refraction, if we could correct only astigmatism at the vertical and horizontal meridians. Later it will undoubtedly be possible to dispense lenses that correct the aniseikonia in all meridians in which it exists. An important question is whether aniseikonia will produce symptoms. Those who introduce this question cite the fact that aniseikonia occurs when one looks obliquely at a page of written matter; that there may be as much as 4 per cent size difference from one side of the page to the other and if symptoms are produced by aniseikonia why would they not be occasioned in such a reading test as this? The answer is probably that they would be produced in individuals who are subject to discomfort from small variations from the normal.

We may ask why one person with a small per cent size difference between the images of the two eyes will not be affected by it, while it will seriously affect another individual. The same question, however, applies to the subject of hypermetropia, for example. How can it be that an individual having, let us assume, ten diopters amplitude of accommodation, may get symptoms from failure to correct one diopter of hypermetrophia, let us say, and that the correction of this will produce comfort, whereas another person with ten diopters amplitude may not suffer from three diopters of hypermetropia. It seems, therefore, as if there is nothing illogical in the fact that aniseikonia of a given amount will cause no symptoms in one individual and still will produce symptoms in another.

A serious technical difficulty is that tests cannot be made by the average ophthalmologist since special instruments and special technic are necessary. The set-up and correcting lenses are expensive so that it is not possible as conditions

RESULTS IN PATIENTS WHO HAVE WORN EIKONIC CORRECTIONS NOT LESS THAN 9 MONTHS

101 REPLIES

		Per Cent
50	Very satisfactory	49.5
33	Moderately satisfactory	32.7
18	Unsatisfactory	17.8
101		100.00

Table 1

GLASSES WORN MORE THAN 2 YEARS

		Per Cent
Number of patients	20	
Very satisfactory	10 or	50
Moderately satisfactory	10 or	50
Unsatisfactory	0	
		100

Table 2

GLASSES WORN 18 TO 24 MONTHS

		Per Cent
Number of patients	35	
Very satisfactory	21 or	60
Moderately satisfactory	8 or	22.86
Unsatisfactory	6 or	17.14
		100.00

Table 3

GLASSES WORN 12 TO 18 MONTHS

		Per Cent
Number of patients	34	
Very satisfactory	15. or	44.12
Moderately satisfactory	11 or	32.35
Unsatisfactory	8 or	23.53
		100.00

Table 4

GLASSES WORN 9 TO 12 MONTHS

		Per Cent
Number of patients	12	
Very satisfactory	4 or	33 1/3
Moderately satisfactory	4 or	33 1/3
Unsatisfactory	4 or	33 1/3
		100

Table 5

now exist to have the test made on all patients upon whom one would desire to have it made. It does not seem feasible to one who has worked with this subject for a good many years that all ophthalmologists should be prepared to make aniseikonic tests. The examination is very time-consuming and is of a meticulous nature that would not be of interest to many ophthalmologists. We have found it impossible to make more than two or three, or at the most, four tests in one day with one technician and one instrument. A special technician* has occupied his full time with this testing in our department for a number of years. Cooperation with an ophthalmologist is essential, because so frequently factors pertaining to the broad aspects of ophthalmology have a bearing on the subject. The psychologic attitude has to be studied; the question of phorias; muscle power, and so on. The general medical consideration of the patient is an element to be weighed seriously in deciding on a prescription for aniseikonia. Not to have an adequate number of instruments for examinations of all those needing them is a serious matter, but to have every ophthalmologist using eikonometers would be hard to conceive. Unless plenty of time is used for the examination the result is not satisfactory. The question of mere measurement of aniseikonia is not sufficient. One must study the tolerance for aniseikonia and the amplitude of aniseikonia before one can be justified in writing a prescription. It would be natural for the busy ophthalmologist to fail to take the necessary time to study all of these points and to prescribe only on the immediate findings and this, in our experience, would not give good results. I am convinced that there is much of value in the study of aniseikonia and the correction of aniseikonia in some patients. I have seen results that are almost incredible and have known many patients who attained comfort, who had never been able to get eye comfort through any other test. I have been very much of a skeptic on the subject, but have finally become convinced that aniseikonia is a matter of real importance. It seems essential that the test should be made available to all patients needing it, and that the expense of it should not be prohibitive. I do not know exactly how these things can be accomplished. It is, of course, to be hoped that the price of the glasses can be reduced and that centers can be set up where the test can be made at not too great expense. There will, of course, have to

*Mr William F. Davis.

be charity clinics as well as pay clinics just as in every branch of medicine. The overhead is heavy. The expense of a technician, of the instrument, and of the glasses all have to be met. At present this is being done in most clinics by the patient who orders glasses. However, this is not entirely satisfactory. In spite of the difficulty, the good results we have obtained at Washington University as illustrated by the first 101 replies to a questionnaire sent out in October of this year to those who had received an eikonic correction not less than nine months previously, as well as the figures from other clinics, indicate that in spite of the difficulty, the subject is well worth pursuing.

THE USE OF HEPARIN IN THE TREATMENT OF THROMBOSIS OF THE CENTRAL VEIN OF THE RETINA*

By RALPH O. RYCHENER, M.D.
Memphis, Tennessee

Heparin is an anticoagulant developed from the discovery by J. McLean[1] in the laboratory of W. H. Howell in 1916, that certain impure heart and liver phosphatides were inhibitory to blood coagulation. As the result of extensive investigations under the direction of Howell[2] (Johns Hopkins), Best[3] (Toronto), Fischer (Copenhagen) and Jorpes[4] (Stockholm) the heparins have been identified as substituted polysaccharides belonging to the class of mucoitin-sulfuric esters. Many varieties and fractionations are possible and purified natural products, including crystalline salts, differ in chemistry and potency. The simplest and most practical assay by Howell, defines a unit as that amount which keeps one cubic centimeter of cat blood fluid for twenty-four hours, at 0°C. Heparin is present in most tissues but the best sources are the liver and lungs.

The experimental work of Ferguson and Glazko[5] in the field of blood coagulation has confirmed the following mechanism: first phase, prothrombin→ thrombin; second phase thrombin + fibrinogen→ fibrin. The conversion of prothrombin to thrombin requires certain activators, the chief being thromboplastic enzyme (trypsin-like), while calcium salts and the phospholipid cephalin are essential co-factors. The clotting reactions, especially in their time relations, are sensitive to physical conditions, including dilution, temperature, hydrogen ion concentration, salt content, "wetting" and other colloidal factors. Special inhibitors are antiprothrombin (first phase) or antithrombic (second phase). The latter may be "immediate" or "progressive." Proteolytic phenomena in blood coagulation do not result in nonprotein nitrogen increase, as in ordinary protein digestion, but probably represent minor degradation and perhaps rearrangement of the significant protein complexes. Crystalline trypsin is (a) thromboplastic (b) thrombinolytic, (c) fibrinolytic, and there is evidence that the natural enzyme of tissue extracts (and plasma) may show all three actions. Clot reaction may be the initial stage of fibrinolysis.

Heparin inhibits the tryptic digestion of casein and also retards the thrombin-destroying action of trypsin and of serum. It is, therefore, antagonistic to "progressive" antithrombins. In purified systems heparin has very little effect on fully-formed thrombin, but the addition of a plasma or serum co-factor (proantithrombin), which is not crystalline albumin, gives marked and prompt inhibition. This resembles the immediate antithrombins, including ferrocyanides.

An equally important heparin effect (Howell, Ferguson) also assisted by a co-factor, is its antiprothrombin action in the first phase of clotting. There are two components: (1) slowing of rate of prothrombin activations due to inhibition of the trypsin-like thromboplastic enzyme and (2) lessening of amount (effectiveness) of thrombin produced. Experimental conditions must be chosen carefully to establish these significant facts.

Heparin is now obtained in commercial quantities from beef lung and is a chemical entity, an acid of the mucoitin-sulfuric type (C_{25} H_{65} O_{30} N_2 S_5). A non-toxic solution of heparin suitable for intravenous injection has been developed by the Connaught Laboratories, University of Toronto, and has been successfully used in over four hundred medical and surgical cases there without a single untoward effect.

Solution of heparin is dispensed as a sterile solution in rubber-stoppered vials which each contain 10,000 units of heparin in 10 c.c. of physiological saline. It has been highly purified by processes which include crystallization with a barium salt. Trikresol 0.3 per cent is added as a preservative. Because of an excessive import duty tax each vial costs about five dollars delivered in Memphis. Another prepara-

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tion, made in the United States,* is biologically standardized so that 1 c. c. will keep 5,000 c. c. of plasma in the liquid state, *in vitro,* for 4 hours at 37°C. Each c. c. contains the equivalent of 10 mg. of highly purified heparin. This product is available in 10 c. c. rubber-capped vials which cost $4.75 delivered in Memphis.

As regards clinical administration and dosage, it is usual for 1 c. c. of the solution of heparin to be added aseptically to 100 c. c. of sterile physiologic saline. This dilute solution is given by intravenous saline drip at whatever rate proves to be required to establish and maintain the clotting time of the patient's blood at about 15 minutes, that is, at two or three times the normal clotting time. Patients differ considerably in their response to heparin. Hence the proper rate for its administration varies from patient to patient. Similarly, the number of days during which it is desirable for the administration to be continued varies from case to case. Ordinarily from 20 to 30 c. c. of solution of heparin are needed daily during the course, usually between seven and ten days.

In addition to all precautions indicated in the administration of biologicals by intravenous saline drip, a special precaution in the use of solution of heparin is avoidance of overdosing. This precaution can be appropriately exercised only, of course, through utilization of facilities and personnel, adequate and competent to insure that administration of the product be properly controlled. No difficulty in this respect has been encountered usually. Schall[6] reported a patient with cavernous sinus thrombosis in whom the coagulation time was prolonged to three hours with resultant massive hematuria. DeTakats[7] states there is no evidence to show that prolongation of the coagulation time of twenty minutes is ever necessary. Under his observation at St. Luke's Hospital, Chicago, where heparin is being used extensively, no thrombus has formed nor has there been a propagation of a pre-existing thrombus while the coagulation time has been kept between ten and twenty minutes.

Heparin has been extensively used in the prophylactic treatment of thrombosis and embolism, blood transfusion and blood analysis. In our special field of ophthalmology it has been employed, without success, in cases of occlusion of the central artery of the retina due to embolism, but encouraging reports indicate that it may have some value in cases of thrombosis of the central vein.

*Liquaemin, Roche-Organon, Inc.

The mechanism of obstruction of the retinal veins by venous thrombosis is, according to Duke-Elder,[8] a complicated process dependent upon events both in the arterial and venous parts of the circulation. The first predisposing factor is a slowing of the blood flow, usually determined by changes in the arteries, so that the entrance of blood is impeded, and in the veins so that its exit is obstructed. The second factor is the formation of a roughened area on the endothelium upon which deposits may collect. Upon this area are deposited cells and fibrin from the relatively stagnant blood stream and a thrombus projects into the lumen of the vein and grows by accretion until the lumen is practically blocked and blood flow almost entirely stopped. Verhoeff[9] insists that endophlebitis with proliferative activity of the endothelium is the real cause of venous obstruction. Following acute infections such as influenza, an acute phlebitis may cause total occlusion. The end result of all these processes is to reduce the circulation to the vanishing point. Total occlusion occurs and the striking fundus picture of engorged tortuous veins imbedded in an edematous retina, accompanied by massive capillary hemorrhages of all sizes and shapes, is produced. These are not restricted to the nerve fiber layer but also involve the nuclear layers, where much damage is done, followed by atrophy. Visual acuity is seriously affected if the occlusion is total; it may be spared in cases of branch blockage but here, too, the macula often suffers, if the temporal branches are involved. Circulation is eventually restored by canalization of the clot and establishment of collateral vessels, but visual results in spontaneous recoveries are usually poor. The most distressing complication, however, is the establishment of hemorrhagic, secondary glaucoma with accompanying pain which yields to no treatment save enucleation. Duke-Elder says that glaucoma occurs in 10 to 20 per cent of cases of complete thrombosis but our personal experience indicates that it occurs in almost 50 per cent of our patients. Several observers have indicated an average onset of increased tension in from 80 to 100 days following occlusion. This complication occurs relatively infrequently in tributary thrombosis. Treatment, in general, has been most unsatisfactory.

In 1936, Holmin and Ploman[10] reported a successful visual result following administration of heparin, which was confirmed soon after by Böstrom and William Olson[11] and Arkle.[12] About twenty cases of complete and partial thrombosis were treated by these continental observ

ers and in ten, full or partial vision was restored. These results were further confirmed by reports from the Mayo Clinic[13] and McDonald[14] of Toronto, and isolated case reports from Ferguson[15] and others have appeared from time to time. Most of the good results have occurred in patients in whom only one branch of the central vein was involved. But occasional patients with complete involvement have had a successful restoration or preservation of vision.

Since 1939, Dr. Ellett and I have had nine patients with complete or partial occlusion of the central vein of the retina who have been subjected to treatment with heparin. There were 6 males and 3 females who ranged in age from 41 to 68, and all showed unmistakable signs of retinal arteriosclerosis. General arterial hypertension was an invariable finding, although the blood pressure was frequently lowered for varying periods. The occlusion was partial in three eyes, total in seven; one man had an involvement of three branches in the right eye and an old total occlusion in the left. Our first patient who had a total involvement of some months duration was treated at our suggestion by his home physician, but as a stable solution of heparin was not available then, he was inadequately treated and he is, therefore, excluded from further consideration.

Of the three cases of partial occlusion, two did very well with a return of normal visual acuity. In one of these, the two superior branches were involved with intense edema of the macula accompanied by some central hemorrhages. In the other, all but the inferior temporal branch of the vein were fully involved but complete resolution occurred. The third patient, a 41-year-old man, should have responded best of all, but six weeks later the fundus picture was unchanged and vision had dropped from 6/6 to 6/12. Repeated efforts to obtain a re-examination in the year following his last observation have been without avail.

Of the six eyes with total occlusion, a man, aged 68, with the typical picture of thrombosis in his only eye improved from 5/60 and J/20 to 6/20 and J/8, two weeks following administration of heparin. Although his visual failure was present for three weeks, he volunteered an improvement in vision on the fourth day of treatment. This improvement was maintained for six months, but nothing more was heard from him for eighteen months when last week he returned to the office in response to my general letter of invitation to all in this series. Whereas previously he had been led by an attendant, he now made the journey by bus from Marianna, Arkansas, to our office by himself. His visual acuity was 6/20 improved to 6/15 + 2 with +1.00 cylinder, axis 180, and an addition of a +3.00 sphere enabled him to read J/3 print slowly. Examination of the fundus showed that the retina was entirely free of hemorrhage save for one minute, round, petechial hemorrhage at the upper border of the macula. All old hemorrhage was absorbed without evidence of any previous disorder except about the fovea, where some cystic degeneration and atrophy occupied the area formerly covered by hemorrhage and exudate. The veins were full, the arterioles slightly contracted and there was evidence of sclerosis at the first vessel crossing of the superior nasal branches. Capillary vascularity was apparent on and below the optic disc. The tension of the eye was 18, Schiotz. The general blood pressure readings were 192/100.

A woman with only one eye suffered total occlusion three weeks before observation. Visual acuity was 1/60 and was unimproved two weeks after beginning therapy. However, her husband reported after six weeks that there was a definite visual improvement. She is crippled and it is difficult for her to get about; as she lives some distance from Memphis, she has not been seen in the past year. The other four eyes made no apparent response, save some temporary improvement in absorption of hemorrhage, and of these, two developed secondary glaucoma and had to be removed. The first had a corneoscleral trephine by Dr. J. C. Adams, Greenwood, Mississippi, the second a paracentesis by Dr. E. C. Ellett, prior to enucleation.

The pathologic examinations as reported by Lieutenant Colonel J. E. Ash of the Army Medical museum were as follows:

Rev. H. G. B., Microscopic.—There is a perforating scar of the cornea at the limbus which incarcerates iris pigment and tissue. Between this and the iris root there is a wide coloboma on the iris. On the other side there are peripheral anterior synechiae, pigment-laden phagocytes in the new-formed angle, and slight scarring of the pupillary margin, with pigment on the lens capsule indicating that posterior synechiae were present. The ciliary processes are hyalinized. The choroid is atrophic and there are small excresences on Bruch's membrane. The retina shows microcystic degeneration and secondary pigmentation at the ora serrata. There is very marked thickening and hyalinization of large and small retinal vessels. One vessel is recanalized and a few chronic inflammatory cells are present in its walls. Near this there is hemorrhage. There are disappearance of ganglion cells and gliosis of the nerve fiber layer around the disc. Posteriorly there are serous exudates in the outer plexiform layer, microcystic degeneration and loss of normal

architecture. The optic disc is cupped, the lamina cribrosa is depressed and there is some atrophy of the optic nerve. In a cross section of the optic nerve the central vessels are seen to be patent and engorged. There is a little acidophili exudate in the anterior vitreous chamber.

The diagnosis is arteriosclerosis, retinal; occlusion and recanalization of the superior branch of the central retinal vein; glaucoma, hemorrhagic; and iridectomy.

Mrs. K. T. A., Microscopic.—The corneal epithelium is very thin and desquamating. Chronic inflammatory cells and polymorphonuclear leukocytes infiltrate the substantia. Hemorrhage fills the anterior chamber and there is a little hemorrhage in the ciliary region. There are broad peripheral anterior synechiae, a vascular membrane on the anterior iris surface, ectropion uveae, hemorrhage and necrosis of the iris. The ciliary body is atrophic, its processes are hyalinized. Polymorphs and some lymphocytes infiltrate the iris. The choroid is thin, particularly anteriorly, and lymphocytes scantily infiltrate it. There is cystic degeneration of the retina at the ora serrata. One subretinal hemorrhage is seen just anterior to the equator and multiple hemorrhages are present in the nerve fiber and outer plexiform layers. Serous exudates are also present in the nerve fiber layer. There is sclerosis of retinal vessels around some of which is seen phagocytized pigment. Large posterior vessels are dilated and engorged. There is considerable disturbance of the normal retinal architecture and slight microcystic degeneration. There is almost complete disappearance of ganglion cells and considerable gliosis of the nerve fiber layer. There is some new blood vessel formation, a little perivascular lymphocytic infiltration, and a few corpora amylaceae in the optic disc. The lamina cribrosa is depressed. The optic nerve enters the eye at an angle with choroid and retinal pigment epithelium extending well out over the nerve on one side. The central artery is engorged. A cross section of the optic nerve shows a crescent of organized clot which enmeshes a few lymphocytes and endothelial cells, in the central vein, but there is still considerable lumen.

The diagnosis is hemorrhagic glaucoma, possibly secondary to thrombosis of the central vein; anterior synechia, and hemorrhage into the anterior chamber.

A critical analysis of this study indicates that in our hands the use of heparin in the treatment of retinal venous thrombosis has not been very encouraging. Two patients of three with tributary blocking did very well, but it may be fairly stated that most such cases recover spontaneously. However, one of the three failed to do so even under heparin so we know complete recovery does not always occur. The man, H. F. B., whose left eye was removed because of secondary glaucoma following total occlusion, at the first examination had a thrombosis of the superior branch of the central vein of the right eye, which within five days extended to include the superior and inferior nasal branches as well. Following heparin therapy, resolution took place so that a clear fundus and a visual acuity of 6/6 and J/1 have been maintained for two years.

Of the six patients with total occlusion, one had an immediate fundus and visual improvement which has been maintained for two years; one had a subjective visual improvement which cannot be verified by examination because of difficulty of transportation, and four were unimproved.

The question may pertinently be asked by the medical nihilist,

"What justification is there for treatment with heparin of an arteriosclerotic patient with venous occlusion?"

The answer is that we are usually unable to determine except by pathologic section whether such occlusions as we see in the fundus are due to a thrombus which plugs the vein, to a sclerotic arteriole which blocks a vein by compression or to an endophlebitis which by proliferative activity causes complete obstruction of the lumen.

Böstrom reasoned that in intravascular thrombosis the thrombus acts as a foreign body producing a tendency to clotting in its neighborhood. If the thrombus fails to grow, the coagulation first retracts, which causes parietal canalization and later the thrombus itself is slowly dissolved by the action of the proteolytic enzymes. According to Jorpes, heparin disappears rapidly from the blood with only a small trace being recovered in the urine. As rapid destruction in the tissues is unlikely, one may presume there is a diffusion through the capillary walls into the tissues which are then heparinized. The blood of the extravasated hemorrhages then remains more liquid and may be resorbed more easily. In Ploman's case, most of the retinal hemorrhages remained the same color until they were resorbed with unusual rapidity.

Therefore, it seems reasonable to give such patients whatever benefit may be obtained from a course of heparin therapy, for as yet no injury has ever been suffered by any patient where the coagulation time was not extended beyond twenty minutes. All of our patients were given a guarded prognosis, in which heparin was described as a drug of unknown value. It could be said that the treatment was harmless, but inconvenient, and the expense of the drug and the proper supervision of its administration in the hospital by the internist and laboratory technician were not minimized. Despite the unpromising outlook, all these patients decided that they would give themselves such opportunity for recovery of vision as might be obtained by this form of therapy.

It is quite true that no permanent effect is achieved on the underlying cause of the occlusion as generalized arteriosclerosis or hyperten-

sion are not altered by such treatment. But we do not deny to the patient suffering from coronary disease, such transient aid as medical science has devised, even though we may feel that the end result is inevitable.

It would help to evaluate the future of heparin in this particular condition if a considerable series of tributary and total venous occlusions could be hospitalized and thoroughly studied from every general medical aspect. The comparison of half the number of such a series adequately treated with heparin to the other half who were not so treated, would give us some premises on which to base our future advice to such patients. It is likely, however, that our knowledge will have to come from such reports as those in this paper.

My impressions gained from our experience with this small series are:

(1) It is useless to administer heparin to patients with total occlusion of long standing.

(2) It should be administered adequately (which means extending the coagulation time to at least fifteen minutes and maintaining it at this level for seven to ten days) to all patients with total occlusions which have been present for three weeks or less.

(3) It is worth consideration in all patients with tributary occlusions and should be definitely administered if the thrombosis appears to be extending to other branches of the central vein.

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DISCUSSION (*Abstract*)

Dr. Albert N. Lemoine, Kansas City, Mo.—We all have experienced the hopelessness of the treatment of thrombosis of the retinal vessels, and any ray of hope any one can give us should be grasped.

Enough experimental and clinical research has been done with heparin to prove its value as an anticoagulant to justify its use in thrombosis of the retinal vein when the patient is seen early enough. It is too much to hope that any of us will ever be so fortunate as to see an arterial occlusion early enough and have the heparin ready to be able to benefit the patient. However, the retinal damage in thrombosis of the vein does not occur nearly so rapidly and we can hope and expect some good results. That is especially true in patients in whom there is only one branch involved. We all have seen patients with a thrombosis of only one branch develop occlusions of other branches, or the central vein, and lose all useful vision or develop a glaucoma and lose the eye. It is impossible to evaluate the benefit to patients with partial occlusion, from the treatment with heparin, because without the treatment they might have lost the eye completely.

The difficulty of the technic of administration and the expense are the main drawbacks; but frequently patients are encountered, and that is especially true in those who have previously lost an eye by such an occlusion, in whom the treatment would be as justified as a glaucoma operation. At least if I were the patient that is the way I would feel about it.

I agree with Dr. Rychener that one should not be deterred from using the treatment because of the associated arteriosclerosis. One can never be sure of the expectancy of these patients. In 1931, I had a patient with a superior branch thrombosis with a very severe arteriosclerosis of the retinal vessels. With medical treatment he retained 20/100 vision in his only eye. He later developed increased intraocular tension that was controlled by myotics. He was a newspaper editor and was able to edit his paper until 1940. The lens is becoming opaque, which accounts for his decreased vision in the past year. Some of these patients seem to go through a vascular crisis, and after they recover from it they seem to become quite stationary for several years.

A report of nine ordinary cases is looked upon as very small, but considering the extremely small number of cases in the literature in which heparin has been used in retinal vein thrombosis, this is a really worthwhile contribution.

Dr. Alvin H. Benz, Chattanooga, Tenn.—The literature contains rather numerous reports on the use of heparin, in conjunction with other chemotherapy, in cases of septic thrombi of the blood vessels, as well as its use in the prevention of thrombi in blood vessel surgery and transfusions. But it is quite barren of reports on the use of heparin in non-infective thrombi. For this reason, Dr. Rychener's report is of particular value at this time.

My experience is limited to two cases, both of which became practically well. Ordinarily this should remove any doubt as to merit. The first was one of cavernous sinus thrombosis from a nasal furuncle. The patient was given 25,000 units daily for the first three days. The clotting time was not determined. More heparin was not given because we did not know that a concentrated solution could be given safely, and 2,500 c.c. of fluids intravenously daily is rather a large amount. He was able to tolerate eleven grams of sulfathiazole by mouth daily and received daily small transfusions starting on the fourth day. He developed a bilateral pneumonia on the seventh day and was also given 20,000 units of staphylococcus antitoxin daily. He rallied and is now well except for a right sixth nerve paralysis.

The other patient had a classical partial occlusion of the central retinal artery with widespread marked edema of the retina, cherry red macula, and near absence of blood in the vessels. A small hemorrhage was seen

from the center of the disk, fanning out over the margin into the retina. Vision was moving objects in the outer temporal field. The patient was given the optimum dosage of heparin with the object of maintaining the clotting time at 12 to 15 minutes. It rose to 27 and 35 minutes at times. Erythrol tetranitrate and ocular massage also were used. After five days, the patient became intolerant of the intravenous and was allowed to go home on vasodilators. Beginning about a week later she showed constant improvement both in vision and fundus picture. At present, 6 weeks after the onset, her vision is 20/40. I believe this patient had a hemorrhage into the sheath of the nerve causing partial occlusion but preventing degeneration of the retina. In both of these cases the part played by the heparin is a matter of conjecture. In the first, an insufficient amount according to any standards was given and in the second, improvement came a week after the drug, and could have occurred quite possibly without its administration.

Heparin, we all know, when it is present in sufficient concentration, prohibits the formation of thrombin from prothrombic substances. This prevents the blood from clotting. Once the clot has been formed, the mechanism of its formation is irreversible and no one has been able to break a thrombus up into its components, fibrinogen, thrombin, and so on. I am unable to find support of the theory that I formally believed, that heparin also acts by dissolving the fibrinous clot. Even if this were partially possible, how could it affect a thrombosis which has been present a week or more and which has undoubtedly undergone considerable fibroblastic proliferation? Therefore, as I see it, it is rational to expect good results from the use of heparin in the prevention of thrombosis and doubtful that it has much effect in doing what we should like to have it do, namely, dissolving the clot.

It is possible that there is a difference between septic and aseptic thrombi. Bacteria as well as degenerating white blood cells give off proteolytic enzymes and it is logical to expect a septic thrombus to undergo lysis more quickly. With sulfathiazole, it is possible to control the infection in the blood stream both before and after lysis has occurred. Septic thrombi also have a greater tendency to enlarge and extend along the lumen of a vessel. Heparin would prevent this. These factors may explain the wonderful results obtainable in cavernous sinus thrombosis by these drugs as against the doubtful ones in thrombosis of the retinal vessels.

Further reports on fresh cases, where the drug may be of value, are necessary and should include dosage, clotting time, and length of treatment.

Dr. O. S. Gibbs, Memphis, Tenn.—I am a pharmacologist. The first practical point about heparin and the biggest and most difficult of all is the price, and, unfortunately, our dear friends in Washington have seen fit to increase this by causing a very large import tax to be put on the best available source, which is from the University of Toronto.

I have had experience with heparin myself very shortly after Dr. Howell announced it, and I made my first batch from dog's liver with instructions which Dr. Howell kindly supplied. It is quite easy to make, but I should like to advise you that all tissues which contain heparin, which are most tissues in the body, also contain very powerful antagonists, and, therefore, very careful purification is necessary, and, moreover, at this time standardization is absolutely imperative.

Various concerns are claiming pure products. These products are pure in the sense that they crystallize fairly sharply in some cases, or that they have a clean solution or some other factor. They should be bought as yet on unitage, and may I say here that the old Howell unit is still probably the most practical, and introduction of new unitage is only confusing the issue at this time.

The second point I would like to raise is that the drug is probably as free as any drug can be from side toxic effects. I have personally given hundreds of injections of heparin to animals in which the blood pressure and respiration were being recorded, and never saw any effect whatever that could not be accounted for by an injection of anything. On the other hand, if you prevent the blood's clotting altogether, then you will get bleeding from any source of injury whatever that may exist, or may start in the body, and excessive heparinization, therefore, has attendant dangers just as hemophilia has. It should, therefore, be avoided.

Heparin is a very good tool indeed. It will prevent clotting. There is no question about that whatever. But it will not, as the previous speakers have already clearly said, undo a clot once it is formed, and any one who may have any ideas on this subject whatever has only to stand behind the laboratory technician trying to clean up a pipette where the blood has clotted. Nothing is known that will clean a blood clot out satisfactorily, and what is true of a glass pipette is still more true in the body. Therefore, we have to consider in practice what use this tool can be to us. It is perfectly obvious that we would like to know all the factors regarding the clotting itself which is not possible at this time. The previous speakers have not raised one practical question in the mechanism of clot formation, namely, the speed of the circulation.

Clots do not form where the circulation is going at a high speed, except under unusual circumstances, as I have shown time and time again in experiments. If we give a cat 25 milligrams of crude heparin, we can stick in an artificial heart and nothing will clot. That is, we can take care of the whole cat, but if we want to measure the blood flow through the salivary gland, which is very slow indeed, at least ten times this amount of heparin is required. In other words, not only in dealing with clotting processes should we try to prevent the blood clotting, but we should do everything we can to keep the circulation as speedy as possible through the damaged part. Various methods, of course, will suggest themselves, but only some of them are feasible, depending upon the part involved. Of course, in dealing with the eye, a very difficult problem is presented because the circulation there is inaccessible.

Now it is absolutely imperative in heparinization as it is with all modern therapy to check the level of the drug. This cannot be done by guessing at the dosage; it cannot be done by guessing at the unitage on the bottle. The only way we can be sure that we have heparinized the case is to test the clotting time regularly. To do that, almost any method may be used. It is very easy to test the clotting time when the clotting time is increased; that is, when the blood is rendered incoagulable. Therefore, almost any method is satisfactory provided one is really familiar with the end point of the particular method. The results of one method cannot be transferred to another.

While this therapy, of course, is still in the experimental stage, I do not think that heparin properly used

is dangerous before clots are formed, and this is one of the many difficulties of the use of the drug, and also why it is relatively unsuccessful. Relatively, I say, because it is so expensive that it is not used in most cases until damage, that is clotting, has occurred. If the drug were very cheap it could be used in cases where one suspected the damage to be likely to occur. While one would undoubtedly waste a lot of heparin, no doubt we would prevent the onset of a great many thrombi and possibly emboli, and I think without any appreciable danger. When the clot is organized, the use of heparin may not do any good, but I do not think it is going to do any harm, because an organized clot stays "put" unless infected. On the other hand, if a clotting process is developing, which, of course, is a relatively quantitative matter, and a small amount of heparin is intermittently given, one may cause weak, friable clots to form which may actually increase the danger of emboli, and, therefore, I would suggest that this point be borne in mind. Either heparinize the patient thoroughly and continuously, or leave him alone.

Finally, I should like to raise one other point which is new, which is interesting, and which has not been mentioned, namely: that when clotting has taken place in vessels, particularly veins, the veins themselves have a deficient blood supply. This causes them to go into spasm, in consequence of which we actually increase the blockage started by the thrombus. This leads us again to the point that all methods in dealing with these processes should involve, whenever possible, increasing the circulation by the use of vasodilators appropriately administered.

ACUTE OTITIS MEDIA AND MASTOIDITIS*

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The subject, acute otitis media and mastoiditis, has been presented innumerable times. I have no new discoveries to reveal to you. The best that I can hope to do is to present some old wine in new bottles. I do not intend to cover the entire subject of acute otitis media and mastoiditis in this short presentation. Instead, I shall treat the subject in rather a hit and miss manner.

Our present day conception of acute suppurative otitis media and mastoiditis is very different from that which was in vogue throughout the United States when I began to study otology. At that time an assistant in the office of a busy otolaryngologist devoted much of his time to the treatment of chronic suppurative otitis media.

Today, thanks to our better knowledge of otology, few cases of chronic otitis media present themselves for treatment. In addition, the mortality from intracranial complications has been definitely lessened, and loss of hearing to improper or inadequate treatment is also less frequent.

One of the most frequent causes of acute suppurative otitis media is the common cold. We are still uninformed as to how this so-called cold affects the ear; moreover, we do not know what changes take place in the ear following its inception. Investigations have not helped us to determine whether the protective action of the ciliated epithelium of the eustachian tube has been injured through the onset of the cold, or if there is a functional change in the local papillary or lymph plexuses of the middle ear. The investigations of Swindle, Spiesman and others show that exposure to drafts may cause vasomotor constrictor changes in the nasal mucous membranes with a lowering of the temperature in those membranes. This reaction is frequently followed by a nasal infection. A similar reflex phenomenon may be the initial stage of a middle ear infection.

That suppurative otitis media and especially mastoiditis occur in epidemics has no doubt been the observation of each and all of you. In Kansas we have had three major epidemics of mastoiditis in the last thirty years. During these epidemics hundreds of mastoid operations were performed; whereas, both in the years preceding and subsequent to the epidemic year very few mastoid operations were necessary. I am stressing the fact that surgical mastoiditis appears in epidemics in order to point out that we cannot be certain of the value of the sulfonamide drugs in preventing surgical mastoiditis until we have had the opportunity of testing them in an epidemic.

After a paracentesis has been performed, some of the factors that decide whether a suppurative otitis media will recover or whether it will later be necessary to perform a mastoid operation are: the virulence and type of the infecting organism, the anatomical structure of the mastoid, whether diploic or pneumatic, the physical condition of the patient, and his own constitutional peculiarity.

The symptoms that warn the attending physician that the infection in the middle ear has spread to the mastoid or its surrounding structures are only variations of those symptoms that normally accompany acute otitis media, namely: fever, pain, discharge and sensitivity of the mastoid to pressure.

(1) Fever variations may occur normally in

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children, but are seen much less frequently in adults. Variations from the normal lytic temperature course, such as a temperature that continues high, or after having returned to normal suddenly becomes elevated or pyemic, must be viewed with considerable uneasiness, especially when associated with unfavorable constitutional symptoms. In patients over 40 years of age suppurative mastoiditis may very often run an asymptomatic course.

(2) Presence of severe pain in the ear or over the mastoid, which continues after paracentesis, should give us concern that the infection has spread beyond the middle ear. Pain behind the eye or immediately around the eye, or in the temple or teeth is suggestive of an extension into the petrosa. This is especially true if it is nocturnal in character. Continuous pain associated with tenderness and edema over the mastoid indicates a suppurative mastoiditis. We must remember, however, that in some cases of mastoiditis, especially in adults, there may be little or no pain. This is especially true in patients of any age in whom the Streptoccoccus mucosus is the causative organism.

(3) If during the course of the disease the discharge continues unabated or increases, or if after becoming scanty it again becomes profuse, or if the secretion becomes scanty and the constitutional symptoms increase, a thorough investigation must be made to determine the presence or absence of a mastoid suppuration. Again, mention must be made of the fact that in a Streptococcus mucosus otitis the discharge may become scanty, and may drain through a small nipple-like projection, or may be entirely absent, and yet destruction in the mastoid may continue. The discharge from a mild case of suppurative otitis usually subsides in from one to three weeks. When it continues from four to eight weeks, either constantly or intermittently, one may feel certain that something is interfering with the normal course of healing. This may be due to the presence of diseased tonsils and adenoids, infected sinuses, poor physical condition of the patient, or to an involvement of the mastoid. Some authorities have advocated operating upon the mastoids of all cases of otitis media in which the discharge has continued for a period of three weeks, asserting that it is safer to operate than to risk the possibility of complications. I do not believe in laying down definite rules relative to the duration of the discharge. Each case should be judged on its own merits.

(4) A tentative diagnosis of suppurative mastoiditis should be made if sensitiveness to pressure over the mastoid process does not recede after the acute phase, or if sensitiveness has left and then recurs later in the disease.

Roentgen ray films are important adjuvants in the diagnosis of suppuration of the mastoid process. With excellent films one can determine the type of cellular structure of the mastoid, the position of the lateral sinus, the presence or absence of zygomatic cells, and the height of the middle fossa.

When the mastoid cells are invaded by a purulent infection, the cell outlines become less distinct and the sigmoid sinus markings become more visible. When serial films are taken, changes in the cells and their septa can be observed. In the coalescent type of infection the septa disappear and abscess cavities make their appearance. In these cases a perisinus abscess can often be discerned and even a destruction of the tegmen frequently may be seen. In the Streptococcus mucosus type of infection, the septa may seem to be intact and yet on operation we may find that the entire mastoid bone is soft and can be removed without effort. The x-ray film is of but little assistance in the hemorrhagic type of mastoiditis, as in these cases there is but little destruction of the cell septa. Both mastoids must be filmed so that a comparison can be made. It must not be forgotten that mastoid changes may not be discernible if one of the sulfonamides has been administered to the patient. In a personal communication, Dr. Law said that under these conditions he did not feel safe in attempting to interpret single plates, but felt somewhat safer when stereoscopic films had been taken.

I shall now present a number of miscellaneous diagnostic factors which at times help in determining the necessity of surgical interference.

(1) When the small cell type of mastoid is infected, sensitivity to pressure is less likely to be present.

(2) A destructive process proceeds more slowly in the small cell type of mastoid but intracranial complications are more likely to occur.

(3) Mastoiditis is more dangerous in the aged but can usually be operated upon with safety at an earlier date than in younger people.

(4) There are cases in which the drum heals completely but suppuration of the mastoid continues without violent symptoms. In my own experience a complete destruction of the mastoid was found in a case of this type, the ear having been free from discharge for two months.

(5) Pains in the ear and mastoid are very fre-

quently caused by a myalgia of the neck muscles. This pain is often confused with that produced by otitis media and mastoiditis.

When any of the following indications are present, mastoid surgery is usually imperative:

(1) The formation of a fistula through the skin which may be traced into the mastoid process.

(2) A subperiosteal abscess over the mastoid or tympanozygomatic region.

(3) The formation of a Bezold abscess.

(4) A large amount of infiltration over the mastoid area, with tenderness or pressure.

(5) A definite drooping of the posterior superior canal wall.

(6) A severe hemorrhagic nephritis which does not respond to ordinary treatment, and which is associated with a severe otitis media.

(7) Symptoms of intracranial complications.

(8) Symptoms of petrositis.

TREATMENT

Local Therapy.—The underlying philosophy of local therapy is to establish and promote drainage and to keep the auditory canal as free from secretion as possible. A culture should be taken at the time of paracentesis or as soon thereafter as possible.

General Therapy.—When constitutional, local, nasopharyngeal or tonsillar manifestations are marked, it is advisable to put the patient to bed, force fluids and institute such constitutional therapy as is indicated.

Sulfonamides.—An entire paper could be devoted to this phase of the subject. Time will not allow me to do more than touch a few of its high spots. There is as yet no unanimity of opinion in reference either to the value of the sulfonamides in the prevention of surgical mastoiditis or as to how and when they should be used. However, most investigators and clinicians agree that these drugs have little or no value when destruction of the mastoid cells has taken place. It is also a well established fact that sulfonamides frequently mask symptoms that are indicative of intracranial complications. Finally, it has been shown that the sulfonamides in some way change the normal roentgen ray picture in mastoiditis so that this otherwise important adjuvant in diagnosis is rendered more or less valueless.

There is a school of thought that believes that a sulfonamide should be given as soon as possible after the onset of an acute infection and should be continued for at least ten days after all symptoms have subsided. On the other hand, Champ Lyons and his associates have shown that the action of the sulfonamides is rendered ineffective unless the patient has been able to develop antibodies. They therefore advise that 24-48 hours be allowed to elapse between the onset of the patient's infection and the administration of the sulfonamides. They also have shown that much better results can be obtained where the sulfonamides are combined with immune serum therapy.

As a rule I do not prescribe the sulfonamides to my ambulatory patients. I insist on a preliminary red, white and hemoglobin determination which should be repeated at least every 48 hours. The initial dosage that is necessary for a given case depends in a measure on the severity of the infection. The subsequent dosage depends not so much on the age and weight of the patient as on the sulfonamide blood level. I no longer have the courage to give these drugs for the prescribed ten days following the remission of symptoms, due to the fact that I have had three cases in which otitic sepsis developed which did not become manifest until the drug was withdrawn. In fact, I have no definite rule as to how long they should be continued. The drug is discontinued when surgical interference is decided upon.

Operative Therapy.—No hard and fast rules can be devised either as to the necessity of an operation or as to the opportune time to perform it. Each case presents an individual problem. Our final decision will depend upon our clinical experience, judgment, and quite often on intuition.

PLEUROPULMONARY TULAREMIA*

REPORT OF A CASE WITH RECOVERY

By J. Owen Finney, M.D., F.A.C.P.
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In 1920 Francis noted the similarity between a plague-like disease of rodents and a human infection called "deer fly fever."[1] He named the disease tularemia after the causative organism, Pasteurella tularensis. Since his observation the disease has been found to be protean in its manifestations. It is now known to involve organs other than those in which the pathologic process

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*From the Guice-Morgan Clinic.

was initially recognized in humans and the clinical picture frequently varies from the neatly designed pattern suggested by early contributors.

Intrathoracic manifestations of tularemia in humans were first demonstrated in 1924 by Verbrycke[2] who, in doing an autopsy on a patient dying with the disease, observed areas of caseous necrosis in the lung and a pleural effusion of straw colored fluid. In 1928 Simpson[3] interpreted certain lung findings in a patient as being areas of tularemic consolidation rather than areas of nonspecific bronchopneumonia. P. tularensis was recovered from the sputum by Bunker and Smith[4] in 1928.

The rarity of reported instances in which the lung and more especially the pleura have been involved in tularemia prompts the report of the following case.

CASE REPORT

J. C., a 26-year-old white married male, a textile worker, was admitted to the Holy Name of Jesus Hospital July 22, 1941. He was delirious at the time and the history was obtained from relatives. He had been in excellent health until ten days before admission, when there had been a sudden onset characterized by a chill followed by high fever. Actual chills had recurred on several occasions during the succeeding three or four days and the additional complaint of chest pain was noted. High fever continued and delirium was present at intervals, but little else of interest was recalled until three days prior to registration, when cough developed. This was associated with marked pain in the right lower chest that was accentuated by inspiration. The cough had remained non-productive. No rash had developed. He drank well water and spring water at times but had been immunized against typhoid fever on numerous occasions. No contact with rats was known. He had not been hunting in two years and had not recently handled a rabbit or squirrel. He was in contact with dogs frequently. He had been given sulfathiazole by his family physician since shortly after the onset of the present illness.

The past history revealed that he had not had a previous febrile illness aside from the usual exanthematous disease of childhood.

The personal and family histories were irrelevant.

Physical Examination.—The physical examination revealed a well developed and fairly well nourished delirious white male. The temperature was 104° F. (r), the pulse 112 per minute and the respirations 40 per minute. The significant positive findings were confined to the chest. Expansion of the right chest was sharply limited while that of the left appeared normal. Tactile fremitus was diminished to absent from the scapula angle to the base on the right. Over this same area the percussion note changed from dull to flat from above downward and the breath and voice sounds were diminished to absent. No friction rub or rales were heard. The left lung was clear. There was some spasm of the muscles of the right upper abdominal quadrant. The skin and mucous membranes were normal except for evidence of dehydration.

The working diagnosis following physical examination was: (1) lobar pneumonia (central), right lower lobe? (2) tuberculous pleurisy with effusion? A note was made that the pulse was unusually good for a patient so very sick in general appearance from pneumonia and that he looked sicker than the average one with a tuberculous pleural effusion.

Laboratory.—Laboratory study on admission revealed a normal urine. The red blood cell count (4,450,000 per cu. mm.) and the hemoglobin (14 grams) were normal. The white blood cells numbered 16,350 per cu. mm., of which 83 per cent were polymorphonuclear neutrophils. Fluid from the right pleural space was examined after each of four different aspirations and the total cell count varied from 250 per cu. mm. to 3,200 per cu. mm. Lymphocytes predominated consistently in the differential study. The specific gravity varied from 1.016 to 1.020. No tubercle bacilli were found. Through an error no agglutination reactions were done on the fluid. A blood culture done on the day of admission was reported sterile after 48 hours. Blood agglutination reactions done on the day of admission were positive for B. typhosus and B. paratyphosus A and B in dilution of 1:100 and were negative for B. melitensis and B. abortus. The blood was not agglutinated against P. tularensis until the sixth day after admission when it was found to be positive in dilution of 1:320. The titer rapidly rose to reach a peak of 1:5,280 the fifth week after onset of the illness.

Roentgenologic Report.—An admission chest film was reported to show areas of increased density throughout the right lung field with thickening of the interlobar septum between the upper and middle lobes and thickening of the pleura at the right base. The left lung field, heart and aorta were normal (*Fig. 1*).

Course.—During the first week in the hospital, or third week of illness, the temperature varied from 103 to 105° F. and the patient was delirious most of the time. The pulse remained strikingly good. Signs of consolidation over small areas of the right lower lobe were first detected clinically during this week and these persisted

Fig. 1
X-ray film of chest taken 8-22-41. Note pleural and parenchymal involvement.

for ten to twelve days. During the second week in the hospital the temperature began to fall by lysis and on the twenty-seventh day of the illness the temperature, pulse and respirations were normal. Aspiration of the right chest was done on four occasions and a total of 650 c. c. of straw-colored fluid was obtained. He was discharged from the hospital August 8, 1941. Marked asthenia persisted until the latter part of December, 1941, when for the first time he felt well enough to return to his work. A recent fluoroscopic examination of the chest revealed evidence of pleural thickening on the right, but the parenchyma was clear throughout.

Treatment.—Treatment consisted of a trial with large doses of sulfanilamide and later with sulfathiazole, neither of which appeared to alter the course of the disease. No antitularemic serum was administered. One blood transfusion was given. It should be mentioned that no medication of any kind had been given for four days just preceding the initial significant fall in temperature.

DISCUSSION

The literature concerning pleuropulmonary tularemia has recently been excellently reviewed by Blackford and Casey.[5] The chief purpose of this article is to add another instance of the occurrence of pleural effusion to the 43 cited by them in their exhaustive study. Their search yielded a total of only 160 cases in which parenchymal involvement had been encountered clinically or at autopsy. These authors point out that pleuropulmonary invasion by P. tularensis is unquestionably much more common than reported instances would indicate.

Tularemia in humans occurs as a result of handling animals, such as rabbits or squirrels, that have the disease, or from the bite of a tick.[6] The wood tick, Dermacentor andersoni, and the American dog tick, Dermacentor variabilis, are the best known carriers.

Pleuropulmonary tularemia occurs most commonly in the so-called typhoid type of tularemia. In this type there is rarely a lesion of the skin or mucous membrane. The absence of such a lesion is said to indicate a virulent infection with progress so rapid that no opportunity is afforded for local abscess formation.

The pathologic process of tularemia is characteristically one of necrosis. The microscopic picture is similar to a caseous focus of tuberculosis. For this reason, among others, differentiation between the two diseases is at times difficult. The difficulty is accentuated in the presence of pleural effusion which may be an outstanding manifestation of either disease.

The diagnosis of pleuropulmonary tularemia depends first, in most instances, on having the possibility in mind, for a lesion of the skin or mucous membrane is usually not present to suggest such an infection. After tularemia is considered various procedures are then in order to test the concept. During the first few days a positive blood culture may be obtained if media fortified with cystine is used. This failing, one may inoculate a guinea pig with blood from the patient. An animal inoculated with infected blood usually dies in from two to four days and autopsy will reveal characteristic lesions of the liver, lungs and lymph nodes. Culture or animal inoculation with material from lesions of the skin or mucous membranes may give conclusive evidence when such lesions are present. In cases of pneumonia, culture and animal inoculation with material obtained on lung puncture are reported to be fruitful diagnostic procedures.[7]

A positive skin test with P. tularensis antigen may be obtained. After the tenth or twelfth day of illness agglutination reaction of the blood serum is, as a rule, positive against P. tularensis and the titer will be found to increase from this time on up to the fifth or sixth week after onset. This progressive increase is important in view of the fact that individuals who have had tularemia retain a fair titer, often up to 1:640, throughout life and the change in degree rules out an antecedent infection as being responsible for the serologic reaction.

Clinically and roentgenologically tularemic pneumonia has no unique characteristics. Cough with chest pain and an accelerated respiratory rate are usually present. The cough is productive in only half the cases. Clinical examination of the chest reveals findings consistent with patchy consolidation and this may persist for from ten days to six weeks. Generalized bronchitis may precede parenchymal involvement and may run a course without subsequent parenchymal extension. An occasional case will progress to abscess formation. High fever is present from two to four weeks; at times longer. Roentgenologic evidence of consolidation may occur from a few days to three weeks after onset of the general infection. In certain cases an x-ray film will reveal discrete nodular areas of increased density in the lung fields suggesting tuberculous foci.

Tularemic pleurisy with effusion is almost universally associated with a concomitant pneumonic process. The amount of fluid varies from a few cubic centimeters to three liters or more and its specific gravity and cellular content definitely classify it as an exudate. Fluids that have been tested have been found to agglutinate P.

tularensis in approximately the same dilution as the blood of the respective individuals. A few cases of spontaneous pneumothorax have been reported.[5] These were thought to have been secondary to the rupture of a pleural abscess.

From these described manifestations of tularemia one may readily see that it should be considered regularly in the differential diagnosis when one encounters any bizarre febrile intrathoracic process.

The prognosis of tularemia in general is good. The mortality rate is reported to be from 3 to 5 per cent.[7] However, patients with pulmonary involvement do not share this favorable outlook, as the incidence of pneumonia is reported to be from 40 to 65 per cent in cases that terminate fatally.[5 7]

At the present time serotherapy and chemotherapy are both recommended as effective therapeutic measures in the treatment of tularemia. The report of Foshay[8] which is concerned with the treatment of 600 cases with antitularemic serum is convincing as to the value of this method. Curtis[9] reported favorably on the use of sulfanilamide in one case and May[10] cites recovery in a case of late tularemic septicemia following administration of sulfanilamide compounds. Moss and Weilbaecher[7] frequently obtained striking improvement with sulfathiazole in a relatively small series of cases. Regardless of the choice of therapeutic agent the importance of its use early in the course of the disease is uniformly stressed. In the case reported herein, sulfanilamide and sulfathiazole were used without apparent benefit. Serotherapy was not employed.

SUMMARY

A case of tularemia with pneumonia and pleural effusion is reported. Recovery was uneventful. The course of the disease was apparently not affected by the use of sulfanilamide or sulfathiazole. Anti-tularemic serum was not used in treatment.

The extramediastinal intrathoracic manifestations of tularemia are briefly discussed.

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SCALENUS ANTICUS SYNDROME OR CERVICAL FORAMINAL COMPRESSION?*

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In recent years "the clinical syndrome of pain over the shoulder, frequently extending down the arm, without objective evidence of nerve or circulatory disturbance in the extremity"[1] has been called the scalenus anticus syndrome. This basic symptom complex may co-exist with "radiations up the back of the neck, discomfort referred to the region of the seventh cervical spinous process" and pains radiating over the anterior or posterior wall of the thorax. As the name implies, the scalenus anticus muscle has been held responsible for these symptoms, and in the belief that the muscle produces the pathologic condition by its tightness, cutting of the muscle has been recommended and practiced.

The contracture of the muscle is said to produce irritative pressure on the subclavian artery and brachial plexus (a) in the "scalene angle"[2]; (b) on the first portion of the plexus, between the bellies of the spastic anterior and middle scalenes,[3] and (c) on the terminal part of the plexus at the first dorsal rib which is said to be abnormally elevated.[3] The relief obtained by patients on whom scaleniotomy was performed has been considered proof of the theory.[3]

A critical study of the observations in this syndrome reveals a number of discrepancies, chiefly mechanical and neurologic. In addition, clinical observations and the results of nonsurgical procedures lead one to believe that other factors are productive of this symptom complex and that relief can be obtained by simpler therapeutic procedures.

The scalene angle is formed by the junction of two lines representing the scalenus anticus

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muscle and the superior surface of the thorax (*Fig. 1*). The other ends of these two lines are connected by a line representing the cervical spine so that we have a triangle in which two sides are of relatively fixed length, while the third, the muscle line, can change its length. It is obvious that a contracture of the muscle must increase the scalene angle. If, therefore, the syndrome is produced by a shortening of the muscle and the associated flexion of the neck, one cannot attribute the nerve pressure to the scalene angle. Indeed, because of the opening of the angle, pressure is reduced at this point.

The concept that pressure between the bellies of the spastic anterior and middle scalenes causes the nerve irritation is likewise open to question. The plexus runs between these muscles only near their origin where the attachments are separated by the rigid transverse processes. The two muscles do not run parallel to each other so that flexion of the neck cannot produce enough constriction of the nerve to yield the symptoms.

Tension of the plexus over an abnormally placed first rib can produce pain radiating down the arm. If, however, the scalenus anticus should become contracted to a point where the upward pull on the rib causes nerve symptoms, it is physiologic for the patient to relieve that pressure by tilting the head forward. Furthermore, if one studies the radiating pains of the syndrome, one finds that a lesion at this point cannot directly account for all of them. This was observed by one of the proponents of the scalenus theory who offered the explanation that there must be an "overflow" radiation or a sympathetic nerve involvement.[3] But even this explanation is not satisfactory, and a much more direct nerve involvement should be sought proximal to this area.

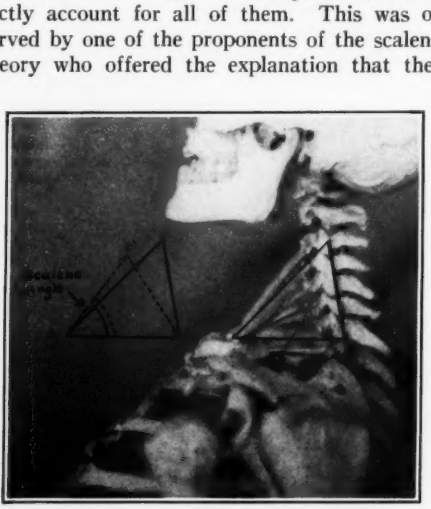

Fig. 1

In this triangle A C represents the scalenus anticus muscle. As the muscle shortens, and the rigid line BC swings on B, the scalene angle becomes greater.

The symptomatic relief reported by patients who have had scaleniotomies is attributed to the elimination of pressure by the muscle. Actually there has not been a uniform sustained relief of the symptoms. I have seen two patients who had recurrence of arm pains after the operation had been performed. Others have reported this experience, too. One is therefore led to ask if the results obtained are not incidental to the operation rather than the direct result of the elimination of muscle pressure. Thus, since the cutting of the muscle allows easier extension of the cervical spine, there is likely to ensue opening of the intervertebral foramina, and this, in turn may relieve pressure on the nerve roots.

Clinically it is worthy of note that though most of the patients have unilateral manifestations of pain, only a small proportion of them have an appreciable lateral tilt of the head. Yet it is to be expected that if only one scalenus anticus is so contracted that it gives nerve irritation, the neck should be flexed laterally. Furthermore, the predominance of the symptom-complex in older people warrants the association of the complaints with illnesses of adult life.

At this point it must be emphasized that the radiating pain syndrome is not a disease entity. It is rather a manifestation of a disease process that affects the nerves of the neck by inflammatory or pressure irritation. Since a number of pathologic conditions may produce the radiations, the syndrome should not be labeled by an etiological name. It is preferable to apply to it a name that describes it anatomically. "Brachialgia" suggests itself as an appropriate name because it indicates the location of the symptoms and does not imply any definite etiologic responsibility.

If the scalenus anticus muscle is to be exonerated, what is most frequently responsible for the arm pains? The lesion must be close enough to the spine to send stimuli through the plexus and yet be able to irritate such nerves as the suprascapularis and thoracalis longus which go on their course without joining the plexus. It must be also sufficiently extensive to permit occasional compression of the roots of the cervical plexus. It is possible to meet these requirements by considering lesions affecting the cervical intervertebral foramina.

The radiation phenomena associated with a single major trauma, such as a fracture of an articular process or a dislocation of a cervical vertebra, are very well known and are definitely attributed to the compression of the nerve in the foramen. In less severe injuries, such as sprains of the cervical spine, one may not be able to give radiographic proof of the origin of the radiation, but it is not difficult to explain them on a similar basis. The effects of prolonged minor traumata, acquired in vertebral malalignment or occupational strains, can likewise produce changes in this location. Finally, arthritis, either of the rheumatoid type with the thickening of the capsule, or the osteo-arthritic type with the bony overgrowths from the articular facets, affords adequate explanation for the radiations. The mechanism for the nerve irritation is quite clear (*Fig. 2*). The nerve roots run through the foramina, the posterior walls of which consist of the joints made by the articular processes. Any displacement of the articular processes forward, and any swelling of the joint at the articular facets, with protrusion of the capsule anteriorly, produce encroachment upon the foramen and reduce the clearance for the nerve. Furthermore, since flexion and extension of the neck are made by the vertebrae "see-sawing" at the articular facets, and since the foramina are anterior to these facets, it must be clear that when the neck is bent forward there is a further reduction in the size of the foramen.

CLINICAL MANIFESTATIONS

The patient may come in complaining of brachialgia that appeared after an injury. The trauma is either that of a major accident, such as landing on the head in an overturned car, or of a simple twist of the neck. Another patient may suggest that the cause was an effort involving prolonged flexion of the neck. Illustrative of this type is the patient who drove an automobile a long distance, riding in a slumped position that made it necessary to carry the head forward to help see over the hood. Or there may have been an arthritis, either of the systemic type with localization in the neck, or of the degenerative type, associated with habitual malposture. Some of the patients may complain entirely of the shoulder, arm or chest pains while others may associate these radiating pains with a stiff or painful neck. As in sciatica, that is, pains radiating down the leg from the lumbar spine, the referred pains may be the major source of discomfort, but may also be subordinated to the spinal pains.

On examination the patients usually show an exaggerated cervico-dorsal angle with the head carried in front of the body. Occasionally one finds a lateral deviation of the head. Muscle spasm is quite uniformly present and is most easily found in the trapezius, which often stands

Fig. 2

(After Spalteholtz). The foramina through which the cervical nerves course are directly in front of the articulations. Flexion of these joints and projections anteriorly from the capsule or processes produce encroachment on the foraminal spaces.

out as an elevated band and generally can be felt as a hypertonic structure. The movements of the cervical spine are restricted. If, as most frequently is the case, the lower cervical spine is involved, the patient may be able to move the head, but careful examination of the lower cervical vertebrae reveals a failure of these cervical articulations to participate in the motion. Pressure directed toward the articular processes produces pain over the affected joints. Frequently passive hyperextension of the vertebrae gives temporary relief, while in others tilting the neck toward the affected side exaggerates the discomfort. In some people elevation of the arm and shoulder girdle with its reduction in pull of the roots in the involved foramina alleviates the symptoms. The sensory phenomena down the arm are not, as a rule, associated with objective changes in the hand. X-ray examinations in the young may fail to show vertebral changes, but in the older groups will reveal osteophytes. These are most easily seen in the lateral views on the bodies of the vertebrae, but can at times be manifested projecting into the foramina if the proper technic is used.

Treatment should be directed to the correction of the etiologic factor but, in addition to this, an effort should be made to relieve the pressure on the nerves at the cervical foramina. Since hyperextension of the neck opens these spaces, mechanical devices that can do this should be considered. Postural treatment in bed, strapping the neck in hyperextension, and the Thomas collar are all useful. Physical therapy is quite helpful. In resistant cases the use of head traction is particularly valuable. After the acute stage is over education of the patient to proper posture with muscle training and rehabilitation are indicated.

CONCLUSIONS

(1) The syndrome of pains radiating to the shoulder and adjacent areas, for which the name "brachialgia" is suggested, is not a disease entity, but is the result of inflammation or pressure on the nerves in the cervical region.

(2) The scalenus anticus muscle is not primarily responsible for the pressure phenomena, and scaleniotomy is not etiologic treatment.

(3) The symptom complex is frequently observed, and can be usually associated with lesions of the cervical spine that produce intervertebral foraminal compression of the nerve roots.

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DISCUSSION (*Abstract*)

Dr. Guy A. Caldwell, New Orleans, La.—I think, after carefully listening to Dr. Nachlas' paper, that he is probably not talking about the same syndrome which Dr. DeBakey and Dr. Gage described some time ago. They were able to relieve the symptoms of this syndrome by section of the scalenus muscle.

These radiating pains have been aptly designated as cervical radicular syndrome. There is a group of patients who continue to have trouble in spite of the treatment outlined by Dr. Nachlas. In these cases, when procaine hydrochloride is injected into the lower part of the muscle as a clinical test, definite relief is obtained for a period of hours and sometimes days and section of the muscle usually gives permanent relief.

The first case of this kind that I saw years ago embarrassed me a great deal. The patient complained of pain radiating down the arm and shoulder and up the side of the neck. I put her to bed with head traction and she was almost completely relieved of symptoms for four or five weeks. However, her symptoms recurred as soon as she was up and about. She became thoroughly disgusted with me and of her own accord consulted Dr. Ochsner, who sectioned the scalenus muscle. The patient was completely relieved.

I wonder how Dr. Nachlas explains the fact that this pain is unilateral. The arthritis, traumatic or otherwise, which might occur in the facets, should be, it seems to me, bilateral in most cases.

What about the circulatory symptoms which are a part of the syndrome that was originally described by Dr. DeBakey and Dr. Ochsner? That would be difficult to explain, it seems to me, on the basis of the pressure on the nerves in the foramina. The patient with a true scalenus syndrome usually obtains temporary relief by elevation of the shoulder girdle and arm on the affected side and this is one of the clinical tests in the diagnosis. This movement, of course, does not change the position of articulation in the neck, and so I wonder, if we are talking about two conditions: one for which Dr. Nachlas has described one treatment and still another which will at times require section of the muscle.

Dr. R. W. Billington, Nashville, Tenn.—I am inclined to agree with Dr. Caldwell that Dr. Nachlas' description of the radicular pain does not fit all of the cases which are usually classified as a typical scalenus anticus syndrome.

Nearly twenty years ago, before this section at the meeting at Chattanooga, I read a paper on low back pain. At that time we were still talking about sacroiliac strains and displacements. By that time I had pretty much lost faith in these as an explanation of our cases of low back pain and particularly those with sciatica. After considerable study of the subject, anatomical and otherwise, I had come to the conclusion that these referred pains into the gluteal region and down the course of the sciatic nerve were the result of nerve root irritation and pressure, that sciatica was a symptom and not a disease. I also called attention to the cases of pains in the neck referred to the shoulder

and arm which I believed to be analogous to these low back cases with sciatica, suggesting that if we should look to the intervertebral foramina and the nerve roots where they emerge through these foramina we would find the cause of the mechanical irritations and pressure, such as arthritis and anomalies, which would account certainly for a large per cent of these cases of brachial neuritis and sciatica.

I do not believe, however, that this explains all the cases of so-called scalenus anticus syndrome, as Dr. Nachlas seems to think.

Dr. Misch Casper, Louisville, Ky.—I probably can offer myself as a patient to add something to this discussion. There seems to be a little difference of opinion as to the modus operandi of the scalenus anticus syndrome. I think we can compromise that difference of opinion. Let us say that the scalenus anticus does have something to do with causing the symptoms. Going further back, what causes a spastic scalenus anticus? I think there would be the crux of the etiology, and perhaps the essayist's remarks that pressure on these nerves has something to do with it are correct.

We observe this condition in surgeons themselves sometimes, who do a great many vaginal hysterectomies and vaginal operations; also in arthritic cervical spines. Certainly cutting the scalenus anticus muscle does relieve it. There are other methods that will relax the scalenus anticus. I wish to call attention especially to short wave diathermy. That has done more to relieve me than anything else I have tried. Also, correcting faulty posture has something to do with improvement. I have even tried, in doing vaginal work, to overcome this spastic condition that results largely from position, by using infra-red light on my cervical spine while I am operating, and it works. So I think here we could compromise and admit that we do have some injury or pressure on the nerve that causes perhaps the spasm of the scalenus anticus muscle, which in turn causes the symptoms and pain.

Dr. Harold B. Boyd, Memphis, Tenn.—Dr. Billington mentioned an analogy between the pains of the shoulder and those of the low back. It is now known that some patients with low back pain have a rupture of the intervertebral disk. Recently Drs. Semmes and Murphey in Memphis have had four patients in whom ruptured intervertebral disks were found in the cervical region at operation. Three of these patients were relieved following the operation. All of them complained of symptoms simulating a so-called scalenus anticus syndrome.

I mention this point in order to call attention to an additional condition which may result in radiating pain in the upper extremity.

Dr. Nachlas (closing).—Dr. Caldwell's suggestion that possibly two separate conditions are being discussed is well taken. To assure myself that this is not the case, I carefully included in my paper, verbatim, a description of the symptoms that are said to constitute the scalenus anticus syndrome. In the written article they will be found in quotations. It must be reemphasized that the syndrome may result from various pathologic conditions: a cervical rib, a tumor, or a herniated disk may produce such radiating pains. It is against the incrimination of the scalenus anticus muscle that this paper is written.

The injection of the muscles that has been reported by various authorities has not proven entirely reliable because it is difficult to circumscribe the anesthesia. In the hands of various investigators secondary sympathetic nervous system involvement has been reported so that obviously the relief of the symptoms cannot be attributed solely to the relaxation of the spastic muscles by means of procaine hydrochloride.

Dr. Caldwell asks: "Why does not arthritis give bilateral symptoms?" I am sure that Dr. Caldwell has seen many arthritic spines whose manifestations have been unilateral. An inspection of skeletons with osteophytes distributed asymmetrically makes it unnecessary to elaborate on this point.

The circulatory phenomena that are mentioned are important. In general they constitute two groups: (1) subjective, such as the numb hand that feels cold; and (2) the group in which objective circulatory defects exist. The first group obviously may constitute part of the picture of a brachialgia. The second group usually results from factors such as aneurysm of the subclavian artery or cervical rib. Such cases constitute the very small percentage of patients who do not fit into the cervical foraminal group.

THE VARICELLIFORM ERUPTION OF KAPOSI*

By ALLEN W. PEPPLE, M.D.
THOMAS W. MURRELL, M.D.
and
RICHARD W. FOWLKES, M.D.
Richmond, Virginia

The following description is taken from Kaposi's[1] "Diseases of the Skin," published in 1895:

"A very alarming complication of eczema larvale infantum, which has come under my observation in several cases, is an acute outbreak of numerous vesicles, partly scattered, partly arranged in groups. The vesicles are as large as a lentil, filled with clear serum, and the majority are umbilicated. They look like varicella vesicles, but undoubtedly do not belong to this class. The integument which has been attacked in this manner now appears still more swollen, even tense. The little patients have high fever (40 C. or more) and are very restless. The vesicles develop very acutely (sometimes over night) in large numbers, and often continue to appear, in successive crops, for three or four days or even a week. Those which appeared first undergo desiccation, rupture, and expose the corium, or they become encrusted and fall off. The largest number of these varicella-like vesicles are found upon the already eczematous skin, but smaller groups appear upon the previously intact skin of the neighborhood, upon the forehead, ears, neck, and even the shoulders and arms.

"The outcome of this peculiar affection always is, in

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*From the Department of Dermatology, Medical College of Virginia.

the majority of instances, favorable; the exposed surfaces are covered with new integument in two or three weeks. In many places pigment patches are left over, or even flat cicatrices. The previously existing eczema changes its character only in so far as it has been influenced by local treatment. In a child *aet.* 6 months, under my observation, death occurred after eclamptic seizures on the sixth day of the disease, when the eruption was everywhere recovering and complete defervescence had ensued.

"I am in doubt with regard to the proper term to apply to this varicella like eruption, which has never been seen by experienced pediatrists, while ten cases have come under my observation. Eczema herpetiforme seems to be a suitable appellation. I am also ignorant of its causation. I suspect, however, that we have to deal with local contagion by a fungus, which finds a suitable soil in the epidermis that has been loosened by the eczema and by its vegetation produces the peculiar eruption. The cases were all so rapid in their course that no opportunity for microscopical examination has hitherto been obtained. The threatening fever is not due to blood infection, but to the local dermatitis."

In 1898, under the title of pustulosis acuta varioliformis, Juliusberg[2] described a fatal case which he said belonged to the group described by Kaposi. He noted the clinical resemblance to variola and varicella. In his opinion this was a pyoderma due to a staphylococcus.

In 1935, Ellis[3] contributed a paper entitled "Eczema Vaccinatum" in which he described the eruption in twins, one of whom died. He suggested that the vaccine virus was the etiologic agent, and was able to demonstrate Guarnieri bodies in sections of skin.

According to Gordon[4] the following criteria must be fulfilled before the virus of vaccinia can be considered the etiologic agent.

(1) The suspected material should produce typical vaccinia on the shaved skin of a rabbit by scarification or intracutaneous inoculation, a control test on the same rabbit at the same time with known vaccinia being done to show that the rabbit is susceptible to vaccinia.

(2) After infection with the virus in question the rabbit should prove immune to a subsequent vaccinia test.

(3) The virus should be neutralized by known antivaccinia serum when brought in contact *in vitro*.

(4) Material containing the virus should give specific complement fixation and flocculation reactions with specific antivaccinia sera.

In addition the following would be confirmatory: (1) inclusion bodies (2) Guarnieri bodies (3) the elementary bodies of vaccinia, now established by the work of Paschen, Ledingham, and others, as the actual morphologic form of the virus of vaccinia.

As far as we know all of these criteria have not yet been fulfilled by any one case.

McLachlan and Gillespie[5] reported an epidemic of sixteen cases in 1936.

In June, 1939, an American series of five cases was reported by King[6].

The following case was seen in St. Phillip Hospital and is reported through the courtesy of the Department of Pediatrics of the Medical College of Virginia:

R. W., a negro infant, aged 17 months, was admitted to the hospital on December 4, 1939, with the following history:

On the morning of December 1, 1939, the mother noticed a "kernel" below the right ear, and by noon the right side of the face was swollen and the right eye closed. On the morning of December 2, 1939, the left side of the face was similarly involved and the child was restless and fretful. The mother added that at this time "pus began to come from the bumps." The past history was irrelevant except that the child had had eczema since he was six weeks old. The patient had never been vaccinated against smallpox.

Physical examination revealed a pustular eruption upon the scalp, face, neck, and extremities. All of the lesions were situated upon eczematous areas except two on the left anterior chest. Unruptured pustules were umbilicated. The roofs of these vesicles were tough and ruptured with difficulty. Many of the lesions on the scalp and face were confluent. The cervical lymph nodes were enlarged. (*Figs. 1* and *2*).

On admission the temperature was 104° F. The leukocyte count on the same day was 22,900 per cubic millimeter. A smear of the blood showed 7 per cent eosinophils. Bacillus diphtheriae was found in smears and cultures from the skin, nose, and throat. This organism was found to be non-virulent. Sweitzer and Ikeda[7] reported positive cultures of Bacillus diphtheriae among many of their cases of variola. The Schick, Wassermann, and tuberculin reactions were all negative.

A smear from an unruptured pustule was stained with Morosow's technic; Paschen's elementary bodies were not present. Some of the pus was rubbed into the scarified cornea of rabbit according to Paul's method. A kerato-conjunctivitis resulted but sections of cornea failed to show inclusion bodies (*Fig. 3*). The scarified skin of rabbit was also inoculated with the pus, but here again sections did not show inclusion bodies. Two unruptured umbilicated vesicles were excised from the chest, and sections of this tissue revealed intra-cytoplasmic inclusion bodies similar to those described by Guarnieri. (*Figs. 4* and *5*).

Case 2.—Mrs. W. A. B., white, aged 28 years, three days prior to her admission to the hospital on March 4, 1939, had a severe headache and backache. A tentative diagnosis of influenza was made by her family physician. A pustular eruption which involved areas of neurodermatitis appeared on March 4, 1939.

The past history reveals that the patient had eczema during childhood and since has had neurodermatitis all of her life. The patient was vaccinated for smallpox at the age of 2. There was a "take" and a small scar marks the site of this vaccination. This patient was

vaccinated again for smallpox when she was 18 years old but this one did not "take." There was no history of exposure to any one recently vaccinated for smallpox. The patient had not been exposed to smallpox or chickenpox.

Fig. 1
Note swollen eyelids, umbilicated vesicles on lids and under chin, and tendency to confluence.

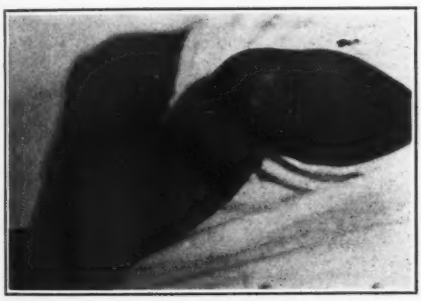

Fig. 2
Leg. Lesions desiccating.

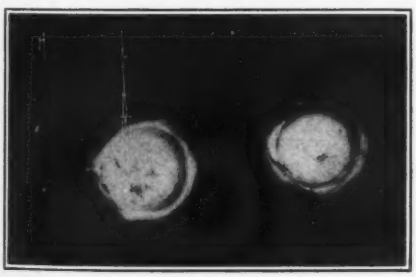

Fig. 3
Left eye inoculated with pus. Right is control.

On physical examination lesions identical with those described in the previous case were observed. The involvement was more general; lesions were present on the trunk. The submaxillary and axillary lymph nodes were enlarged. The maximum temperature was 103° F. The white blood count was 5,900 per cubic millimeter and a smear of the blood showed 8 per cent eosinophils. (*Fig. 6*).

During the period of desiccation crusts were emulsified in saline and rubbed into the scarified cornea of a rabbit. There was no reaction. Some of the crusts were inoculated upon the chorio-allantoic membrane of the chick embryo but no virus was recovered.

Fig. 4
Intracellular edema with many intra-cytoplasmic extra nuclear inclusions.

Fig. 5
Lower down in papilla. Note clear space about inclusions. Magnifications are the same.

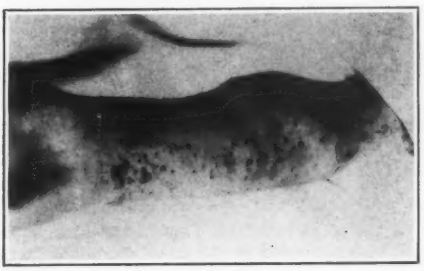

Fig. 6
Anterior surface of forearm; lesions desiccating.

This patient was examined one month ago and there was no evidence of scarring.

Case 3.—R. J., a negro woman, aged 36 years, was seen at the City Home. About seven days prior to her admission the patient was taken with a severe headache and backache. Two or three days later pustules appeared on the eczematous areas. When she was seen the eruption was subsiding but the history corresponded in all respects to that of Kaposi's varicelliform eruption. This patient had had neurodermatitis for most of her life.

All of these three cases made uneventful recoveries.

A brief review of the literature on Kaposi's varicelliform eruption, pustulosis vacciniformis acuta (Juliusberg), and eczema vaccinatum reveals a great deal of controversy in regard to the etiologic agent. Tedder[8] concluded that the three conditions were clinically identical and that the etiologic agent was the virus of vaccinia. The British authors, McLachlan, and Gillespie[5], who had the opportunity of studying an epidemic of 16 cases were unable to find any evidence of a virus. In all of their cases they isolated a streptococcus. In passing, it should be mentioned that tissue was not excised in all of their cases, yet no evidence of inclusion bodies was found in cases where excised tissue was examined.

Brain and Lewis[9] in 1937 were unable to find any evidence of vaccinia virus in their case.

Bettley[10] reported a case in 1940 in which he was unable to find any evidence of virus infection. He said that his patient had been isolated in a hospital 23 days before the eruption appeared. This patient had also been vaccinated successfully before the varicelliform eruption appeared.

Freund[11] obtained a positive Paul test in his case. He was able to find Guarnieri bodies in sections of the rabbit cornea. It is interesting to note that he vaccinated his case successfully with the production of secondary pustules after the varicelliform eruption had cleared.

Buddingh[12] has suggested that the chorio-allantoic of the chick embryo be used for the cultivation of the variola virus. He reported a case of smallpox in which inoculations of the scarified cornea and skin of the rabbit with the original pustular material were negative, but some of the same pus inoculated onto the chick embryo produced pocks, and the elementary bodies of Paschen and the inclusions of Guarnieri were recovered. Goodpasture and his associates[13] believed that Guarnieri bodies were in part composed of Paschen's corpuscles. Ledingham[3] said that the Guarnieri bodies were diagnostic of variola or vaccinia.

SUMMARY

(1) Three cases are reported, two in adults, which may be classified as Kaposi's varicelliform eruption.

(2) In one case intra-cytoplasmic inclusion bodies similar to those of Guarnieri were observed in the epithelium. A kerato-conjunctivitis resulted when Paul's test was done.

(3) Accepting the Guarnieri body as evidence of variola or vaccinia, this lends weight to the belief that Kaposi's varicelliform eruption is a manifestation of vaccinia.

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DISCUSSION (*Abstract*)

Dr. M. F. Engman, Jr., St. Louis, Mo.—We have produced in patients with neurodermatitis a similar eruption by the administration of magnesium. We have in the last few years been studying chronic disseminated neurodermatitis rather intensely by various methods and have found a magnesium deficiency in the skin of these patients. This work has been published. Of course, the presence of this deficiency at once suggested that therapy with magnesium was indicated. We treated four cases in the Barnard Free Skin and Cancer Hospital with magnesium as follows:

Magnesium sulfate was given intravenously, intramuscularly and also in the form of magnesium sulfate baths. In each case the result was the same. The patient suffered an acute exacerbation of the neurodermatitis, developed a fever with some gastro-intestinal symptoms and there appeared over the entire body a pustular eruption. We cultured the pus and found only staphylococcus. When magnesium therapy was stopped, the eruption rapidly cleared up.

Dr. Charles C. Dennie, Kansas City, Mo.—Dr. Morgan and I, about two years ago, had an infant under observation who we thought presented this type of syndrome. The infant was 11 months of age. It had had

eczema since it was six weeks of age. The condition became so serious that we had to hospitalize the child. About the second or third day that it was in the hospital it developed umbilicated follicular lesions on the palms and soles, and on the body. We were unfamiliar with the syndrome of Kaposi and thought we were dealing either with smallpox or chickenpox. We called in the chief of the Pediatric Department for consultation, and at his suggestion vaccinated the child on the only place on its body where it had no eczema. The vesicular eruptions cleared up, and on about the fifth or sixth day the vaccination began to take. Four days after this, the child had vaccina over its entire body from head to foot: eyelids, scalp, palms, soles, everywhere imaginable, and so thick that we could hardly touch the skin without touching one of the lesions.

We did not examine the lesions histologically. We did culture the original lesions, which contained hemolytic staphylococci, but here was a process which disappeared in its entirety before the real vaccina made its appearance. Whether that is any argument for or against the original lesion being vaccina, I do not know, but that is what happened in this case.

Dr. Murrell (closing).—I am proud of Dr. Pepple's work and very humble myself because, frankly, I missed the diagnosis entirely, and he made it. When I first saw the case, the excessive toughness of the umbilicated pustules around the nose made me wonder whether there was any possibility of diphtheria being present. So I suggested that a smear be made for diphtheria, and the laboratory reported it positive. At that point the professor of pediatrics went up in the air. He said he knew it was not diphtheria and insisted upon virulence tests. These were found to be nonvirulent diphtheroids. Dr. Pepple did not include that in this paper. In the literature, I think it was Michelson who found an intercurrent infection of diphtheroids in smallpox. This was a matter of interest, which seemed to have nothing to do with the etiology.

HYPERPARATHYROIDISM*

WITH METASTATIC DEPOSITS IN THE KIDNEYS

By AUGUST A. WERNER, M.D.†
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Hyperparathyroidism is an excessive secretion of the parathyroid glands. Osteitis fibrosa cystica is its most prominent clinical manifestation. Von Recklinghausen,[1] in 1891, was the first to separate osteitis fibrosa from other bone diseases with degeneration and weakness of osseous tissue.

Askanazy,[2] 1904, found a parathyroid adenoma in a case of osteitis fibrosa and he suggested a cause and effect relationship between the parathyroid tumor and the bone disease. Collip, in 1924, in his early experiments on dogs, using extracts of parathyroid glands, found that it was possible to produce what he described as "overdosage phenomena" characterized by hypercalcemia, accompanied by anorexia, vomiting, apathy, drowsiness verging into coma and a failing circulation. Hueper,[3] found calcification in the lungs, kidneys, stomach, heart muscle and the thyroid gland of dogs to which he gave overdosage of parathyroid hormone. Accompanying these changes there were calcium casts in the tubules of the kidneys and thrombi in the small vessels of the myocardium with localized areas of degeneration.

Jaffe, Bodansky and Blair[4] were able to produce extensive bone changes in guinea pigs and puppies by giving relatively large doses of parathormone for a period of three weeks. There was decalcification, softening of the bones and fibrosis which satisfied all of the criteria of osteitis fibrosa.

Mandl,[5] 1926, recognizing symptoms in a patient which might be due to hypersecretion of the parathyroid glands, explored the thyroid area and found a parathyroid tumor, removal of which was followed by remarkable improvement of the patient. Gold,[6] in 1928; Barr, Bulger and Dixon,[7] in March, 1929; Boyd, Milgram and Stearns,[8] in August, 1929, and Quick and Hunsberger,[9] in 1931, reported cases of hyperparathyroidism with a parathyroid tumor. Wilder[10] reported a case of osteitis fibrosa associated with a malignant parathyroid adenoma, removal of which resulted in marked improvement of the patient.

Hunter,[11] in October, 1931, reported that satisfactory proof of hyperparathyroidism existed at that time in thirty-two cases of generalized osteitis fibrosa. In twenty-one of these cases a parathyroid tumor was removed by operation, in two cases two parathyroid tumors were removed and in four cases no parathyroid tumor was found at operation. The remaining five patients were not operated upon, but proof was obtained at necropsy of a parathyroid tumor in three cases and of two parathyroid tumors in one case.

Etiology.—The condition may be produced experimentally by continued overdosage of parathormone. It has been found to exist in the presence of apparently normal glands which are hyperfunctioning. Hyperplasia of the parathyroid glands has been found associated with this condition in some instances. The most frequent cause of the disease is the presence of an ade-

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noma or an adenomatous condition of one or more parathyroid glands.

The condition is not rare. It is two or three times as common in females as it is in males. It is of rare occurrence before the age of 10 years, and is of increased incidence after the age of 20 years, with greatest frequency from 30 to 60 years of age. Its incidence seemingly has no relation to race, heredity, diet or environment.

CLINICAL CHARACTERISTICS

(1) *Abnormalities in Metabolism.*—The most important abnormality in metabolism is that of calcium. It has been proven that parathormone mobilizes calcium from the bones which are the great storehouses of this element in the body. The blood calcium in this disease may range from slightly above normal (average 10.5 mg. per 100 c. c. of blood) to as high as 23.6 mg. as reported by Hunter. A perusal of the literature on this point in reported cases seems to place the blood calcium level at about 14 to 16 mg. per 100 c. c. of blood. However, there is variation of this figure, depending upon the time the blood is taken and whether or not calcium has been administered. With this hypercalcemia there is an increased urinary output of calcium. There is a decreased serum phosphorus level and an increased excretion in the urine.

(2) *Osseous Changes.*—Osteitis fibrosa cystica represents the typical osseous changes. Tumefactions may occur in the bones and roentgenologic examination reveals osseous decalcification with rarefaction, cystic degeneration, mottling, thinning of the cortex and development of fibrotic changes in the cyst walls. Skeletal deformity results and fractures commonly occur from injuries which seem of insufficient violence for their production under normal conditions. Albright, Aub and Bauer[12] in describing the microscopical appearance of the osseous tissue write:

"The bones become porous and filled with osteoclasts. Osteoblasts, laying down new bone, attempt to keep pace with the osteoclasts and are therefore likewise increased. Both these cells are derived from the reticular cells to form the osteoclasts and osteoblasts. Two additional secondary changes may occur. Clefts may develop in the fibrous areas and enlarge until they form multiple cysts with fibrous walls. Secondly, the osteoclasts in certain areas may proliferate to such a degree that they form osteoclastomas (benign tumors of bone tissue with giant cells)."

(3) *Metastatic Calcification.*—Many of these cases show deposits of calcium in various organs and tissues of the body. Hunter reported renal calculi in ten of the cases given in his article. Barr, Bulger and Dixon found large branching stones in the pelves of both kidneys. Albright, Baird, Cope and Bloomberg[13] found renal calculi in twenty-three of eighty-three cases. Dawson and Struthers[14] found calcium infiltration in practically every organ of the body. Hueper, besides producing decalcification of the bones and hypercalcemia in guinea pigs, was able to produce metastatic calcification in the lungs, kidneys, stomach, heart muscle and the thyroid gland.

(4) *Renal.*—Polyuria is frequently present, the patient voiding 2,500 to 3,000 c. c. daily. This may be accompanied by polydipsia. The urine may be alkaline and milky in appearance with a heavy whitish precipitate upon standing. The urinary calcium excretion is increased. The phenolsulphonephthalein test may be decreased due to impairment of function as a result of metastatic deposit of calcium in the parenchymatous structure of the kidneys.

(5) *Diminished Excitability of Nerves and Muscles.*—The condition of the nerves and muscles in hyperparathyroidism is the exact opposite of that found in tetany. The muscles are flabby and weak and because of this and the condition of the bones the patient may be unable to walk. The response to electrical stimulation is greatly reduced.

(6) Pain and tenderness in the muscles and bones are common.

(7) There are apathy, fatigability and drowsiness.

(8) Gastro-intestinal disturbances such as anorexia and periodic severe pain with vomiting. Constipation is the rule.

(9) *Parathyroid Glands.*—Adenomatous tumors of one or more glands are usually found. As has been the experience of investigators with other ductless glands of the body, the size of the tumor does not determine the degree of hyperactivity. Very small adenomata may produce rapid and marked changes in the body, and vice versa. Again, no tumor may be found, the glands may appear normal, or show only what appears to be a generalized hypertrophy or hyperplasia.

Osteitis fibrosa must be differentiated from such bone diseases as fragilitas ossium, osteomalacia, osteitis deformans, carcinomatous metastases, myeloma, abscesses and tuberculous and syphilitic osseous involvement.

REPORT OF CASE

The following case shows marked decalcification of the bones, with metastatic deposits of calcium in both kidneys and their pelves. This case was brought to the attention of the author just after the death of the patient. A correct diagnosis was not made, although the evidence for the presence of a parathyroid tumor was quite obvious.

The patient, a widowed white woman, 62 years of age, entered a hospital January 2, 1932. At that time she complained of frequency of urination with pain and burning. There was pain in the pelvis which would radiate to the lumbar region of the back. At times she would have nausea, but no vomiting. These symptoms had been present for two years. There was a loss of thirty pounds in weight during that time. Constipation had been obstinate for years.

Past History.—She had measles, mumps, chickenpox and pertussis in childhood and pneumonia at the age of 16 years.

Family History.—The husband is living and well. She had two sets of twins and one single birth. There was one miscarriage. The father died at 84 years of age of a kidney condition. The mother is living and well at 77 years of age. Three brothers and four sisters are living and well.

Physical Examination.—The patient appears old, is emaciated, but apparently not very ill. She is almost bald, except for a fine fuzz on the scalp. The pubic and axillary hair is abundant. She is deaf in the right ear and almost deaf in the left ear. She has pigeon-breast, the chest is hyperresonant and the breathing is emphysematous throughout. There is slight increase in cardiac dullness, the pulse is 70 and the blood pressure is 170/90. Except for tenderness over both renal regions and a firm palpable mass in the right lumbar region, the abdomen is negative.

Laboratory Examinations.—The hemoglobin is 90 per cent; red cells, 4,230,000; white cells, 8,000. Wassermann and Kahn tests (twice) are negative. Nonprotein nitrogen is 18.75 per 100 c. c. of blood.

The urine is cloudy, reaction acid; albumin, negative; sugar, negative; there are many pus cells and some granular casts. Upon bilateral catheterization the left kidney shows some red cells and occasional pus cells. The right has many pus cells. Phenolsulphonephthalein from the left is 3 per cent in ten minutes; right, 3.5 per cent in ten minutes. Both specimens show a growth of B. coli.

A flat plate of the kidney regions shows a large opacity at the level of the third right lumbar vertebra. Evidently there is a stone which is palpable on careful examination.

X-Ray Report.—The left catheter extends to the inferior border of the third lumbar vertebra. The right catheter ascends to the superior border of the third lumbar vertebra. Opaque medium shows a slightly dilated left kidney pelvis. There is a large opacity in the right kidney pyelogram. The catheter extends into the right kidney pelvis to the side of the stone. There is a large kink in the right catheter, just below the kidney pelvis. The right kidney pelvis lies at the level of the brim of the pelvis. The lower pole of the left kidney is at the brim of the pelvis. There is a large stone in the right kidney.

A right nephrotomy was done and a large stone was removed. The patient recovered and was discharged thirty-eight days after the operation.

Five years later the patient was readmitted to the hospital. Since the original operation she has had a dull ache more or less constantly in the right side, with intermittent attacks of sharp pain in this region which radiate to the labia. Three months previous to this

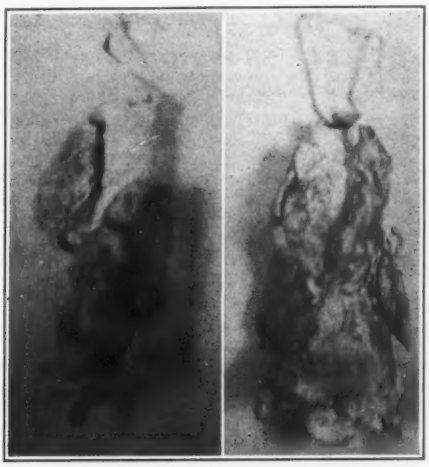

Fig. 1

(Left) Note the large parathyroid tumor posterior to the right upper pole of the thyroid gland, at the side of the trachea.

(Right) Note the same enlarged parathyroid gland as shown in a rear view.

Fig. 2

Note decalcification of the skull.

readmission to the hospital, she had hematuria for three days. One month previously, she had a very severe attack of right lumbar pain, accompanied by nausea and profuse perspiration. Since then she has felt weak and has had malaise. During the last month there has been frequency of urination and burning, especially at night. The urine is cloudy and full of shreds.

The urine is turbid, the specific gravity is 1.014, the reaction is acid, glucose is negative and albumin is two plus. Microscopic examination reveals many pus cells and staphylococci and rod-shaped bacilli.

Bilaterally catheterized specimens show appearance of PSP on the right side in thirteen minutes and on the left in eight minutes. The fifteen minute output on the right side was 1 per cent and on the left 5 per cent.

Nonprotein nitrogen is 26 mg., calcium 10.5 mg., and phosphorus 2.7 mg.

Hemoglobin is 90 per cent, white cells, 9,800.

X-Ray Report.—There are opacities in the region of both kidney pelves. The kidney shadows appear to be unusually low, especially the one on the right. After injection of opaque solution into both ureters the solution was seen to enter the right kidney pelvis surrounding the stone. Only the upper major and minor calices could be visualized. There was marked tortuosity of the right ureter and moderate dilatation of this structure. Only a small amount of opaque solution was seen in the lower portion of the left ureter and none was seen in the upper half of this ureter or in the left kidney pelvis. Examination of the long bones on the left side of the body and of the skull fails to reveal evidence of cystic bone changes or unusual calcium absorption or deposit. The skull reveals very definite trabeculation, a formation resembling changes sometimes seen in parathyroid disease. The changes, however, are not pronounced enough or definite enough to allow a diagnosis of parathyroid disease to be made. (Evidently these roentgenograms were misinterpreted.—AUTHOR).

A second operation was done on the right kidney. The time since the first operation was five years. A pyelophlebotomy and nephropexy were done. The right kidney is at the brim of the pelvis and is held to surrounding structures by dense adhesions. The ureter has an "S" shaped kink just below the right kidney. There is a smooth stone the size of a small lemon in the kidney pelvis. The pelvis is very much dilated but there

Fig. 3
Note decalcification of the humerus.

Fig. 4
Note decalcification of the upper part of the femur.

is a fair amount of kidney cortex. This stone was removed. There seem to be no urethral calculi.

The patient died two days following this operation.

Postmortem Report.—Both pleural cavities contained over a liter of clear serous fluid. The heart was moderately enlarged and the coronary vessels were tortuous and showed intimal thickening. The lower part of both lower lobes was collapsed and atelectatic. The peritoneal cavity was filled with cloudy, seropurulent fluid. The peritoneal surfaces were reddened and injected, but no fibrin or adhesions were present. The liver, pancreas, spleen and the gastro-intestinal tract were negative.

The kidneys were about the same size, and together they weighed 340 grams. The left kidney was filled with stones which could be palpated through its greatly widened calyces and pelvis. The subcapsular surface was smooth and pale. Sections showed the kidney substance to be greatly narrowed. The pyramids were blunted and the calyces and pelvis were enormously enlarged and completely filled with faceted stones. Some of these were quite large, but others were small and fitted in the crevices by faceted surfaces between the larger stones. The ureter below the ureteropelves junction was not widened and was lined with smooth mucosa. The mucosa of the pelvis and calyces was converted into a necrotic, gray, leathery surface.

The right kidney was huge, the pelvis and calyces were dilated and the renal substance showed the loss of substance as was found in the left kidney. No stones were found in the right kidney. (Two operations were done on this kidney.)

The adrenals and the thyroid were grossly normal.

Parathyroid Bodies.—For the purpose of studying the parathyroid bodies the larynx, trachea, esophagus and thyroid were removed intact with the surrounding tissues from the level of the upper margin of the thyroid cartilage.

On the left a small parathyroid was found in the alveolar tissue, adherent to the capsule posteriorly at the level of the junction of the upper and middle thirds of this lobe of the thyroid and another was found below at the margin of the lower pole. The superior body measured 8 x 4 x 2 mm. and the inferior 6 x 4 x 2 mm. They were composed of firm brownish tissue and the cut surfaces were uniform in consistency. The superior gland on the right side could not be found and on the chance that the nodule adherent to the thyroid contained parathyroid, a section was taken, marked (X). Lying on the posterior surface of the trachea to the right of the mid-line and pushing the esophagus to the left was a large oblong structure having a definite capsule which was scarred slightly and ruptured on its lateral surface. From the point of rupture a little brownish serous fluid exuded from the center of the mass. Section showed the tumor to be composed of brown, rather soft cellular tissue containing a cystic, apparently degenerated area in its center and lower portion. The tissue did not extend into any of the surrounding structure. The inferior thyroid artery was adherent to the upper pole of the tumor where it gave off many branches supplying the tumor and then passed to the lower pole of the right lobe of the thyroid. The recurrent laryngeal nerve passed anteriorly to this tumor, apparently supplying it with branches and continuing posterior to the right lobe of the thyroid to enter the substance of the larynx above.

A block was taken from the center of tumor and designated (T). The tumor measured 42 x 19 mm. in its greatest diameters.

Microscopic examination of the parathyroid glands, except the tumor, showed them to be comparatively normal.

A section of the parathyroid tumor showed predominantly two types of cells. The greater bulk of the gland was composed of large cells with abundant hyaline oxyphilic cytoplasm and small dark, almost pyknotic nuclei. These were arranged in anastomosing cords and strands, two cells in width for the most part. Vascular stroma was fairly abundant, but there were wide interstitial spaces which were perfectly clear or at the most contained only a little amorphous precipitate. The other type of cell was smaller, with a neutral frothy cytoplasm and a larger nucleus which was slightly reticular. These were arranged in the same manner as described above. However, more alveoli were present in this portion of the gland and there was less interstitial clear space in this area. These two types of cells were apparently the oxyphil cell and the chief cell respectively as described by Castleman and Mallory.[15]

Except for some vascuolization of the cortex cells, the adrenals and the thyroid were microscopically normal.

Fig. 5

Note the marked decalcification of the pelvic bones and the lumbar vertebrae. The shadows in the renal areas are caused by stones and opaque fluid.

This case convincingly presents the effect of hyperparathyroidism (parathyroid tumor) in causing decalcification of the skeleton, with

metastatic deposit of the calcium in the kidneys and their pelves. Some cases have been reported in which calcium deposits were found in almost all tissues of the body. It also emphasizes the importance of keeping the possibility of hyperparathyroidism (or tumor) in mind when calcium deposits are found in any part of the body, especially in the kidneys and associated structures.

The serum calcium was 10.5 mg. (normal). The phosphorus was 2.7 mg. It is possible that the report of a normal blood calcium may have misled the attending physicians as to the true nature of the condition. Evidently the report on the condition of the bones was very wrong, which again would mislead the physicians. This case emphasizes the necessity for correct laboratory technic and for correct interpretation of roentgenograms and laboratory reports. Also, that the physician should investigate each case personally and not depend wholly upon the work of inexperienced interns or others. Every physician should be capable of interpreting roentgenograms and should view them personally, regardless of the fact that a competent roentgenologist has made a report. No report can impress the picture and nature of pathology so understandably and convincingly upon the physician as a personal observation of the condition can.

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PLASTIC OPERATIONS IN UROLOGIC SURGERY*

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It is obviously impossible to cover the entire subject of plastic surgery as applied to the urological organs in the time allotted to me for this presentation; therefore, I shall merely mention some of the operations involved and describe more fully a few of the procedures which are original with us.

THE EXTERNAL GENITALIA

The external genitalia are particularly subject to developmental deficiencies. Hypospadias, of varying degrees, is relatively common in both male and female subjects. Epispadias is found more infrequently in both sexes. We have seen one amazing case in the female, in which, at birth, there was no orifice whatever in the region of the perineum. This will be described more fully later.

Epispadias.—The surgical correction of epispadias has been fairly well standardized by the operation of Young, which has been fully described elsewhere.

Hypospadias.—Our own operation for the cure of penoscrotal hypospadias has proven very satisfactory in a series of some 50 operations, so that it, also, has become widely and successfully used. A recent improvement is the development of a flexible nonbreakable wire which has been attached to an atraumatic needle. This has been in use for some months and has proven most efficient. The operative procedure is as follows:

At the first operation, the urinary stream is diverted by means of a suprapubic cystostomy, and a tube is formed of scrotal integument. Probably the most important single step in multiple-stage operations for the correction of penile and urethral anomalies is the operative diversion

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*From the Department of Urology (James Buchanan Brady Foundation) of the New York Hospital.

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of the urine. We have found suprapubic cystostomy, using a double suction tube connected with suitable suction apparatus, preferable to drainage through a perineal wound since, in the latter, there is a tendency for urine to pass along the tube into the urethra and thence to the operative site. Having diverted the urine, the next step is to construct a new urethra, which we do from scrotal integument. An inch-wide strip of scrotal skin, extending from the anomalous opening laterally and either downward or upward as far as necessary to secure a piece about an inch longer than the penis, is partially freed by undercutting each edge slightly, and a tube is constructed by approximating the edges over a catheter with interrupted mattress sutures of malleable wire. The edges of the remaining scrotal tissue are then sutured together, so that at the end of this first stage there is an attached tube of scrotal integument, the new urethra, buried in the scrotum. As the end of the tube tends to constrict, a tension suture is threaded into the tube and attached to the suprapubic incision and to the meatus of the new urethra, remaining in position until the final stage of the operation.

The scarred gutter of the distal portion of the anomalous urethra is next dissected away; the deformed penis is relieved of the scar tissue which causes a congenital chordee, and the glans is split into two parts. The cut edges of the penile wound are sutured to the two edges of the scrotal wound, and the glans penis is affixed to the urethra in such a manner that the end of the tube extends for an inch beyond the end of the penis. This excess is required so that any increase in the length of the penis during erection will be accommodated by the urethra, which in the quiescent state of the penis is too long.

Finally, the urethra is freed from its bed. After remaining in position long enough for the new tube to become firmly fixed to the penis (usually from 10 to 14 days), the entire structure is dissected free from the scrotal bed with ample scrotal tissue to cover the freed urethra without pressure. The raw edges of both the penis and the scrotum are repaired. After healing occurs, the operation is completed by allowing the suprapubic wound to heal.

A Human Cloaca.—A most unusual patient, a young girl with a cloaca, was operated upon by us and cured. This interesting case is described in some detail.

M. P., a 9-year-old girl, entered the hospital in June, 1938, with a congenital deformity in the form of a cloaca and inability to control evacuation from it. There had been an appendectomy and a colostomy for obstruction, but no operation upon the cloaca. The surgeon reported that he had found normal female pelvic organs. The family history was negative.

Physical examination showed a healthy, normal girl except for the examination of the abdomen, external genitalia and anus. There was a well-healed right rectus incision and colostomy scar. The recti muscles were separated in the lower abdomen, forming an inverted V with a separated pubis as the base. A soft mass, the bladder, filled this V. The pubes were separated about 7 cm., with a fibrous band across the separation. A bifid clitoris lay between the anterior ends of the two widely separated labia majora. No urethral, vaginal or anal meatus was found. In the space between the labia were two openings, each about 1 cm. in diameter and about 2 cm. apart, from which exuded a mixture of urine and feces. There was no sphincteric action to either opening.

Intravenous pyelograms revealed a normal upper urinary tract, but both the pyelograms and repeated attempts at cystoscopy failed to disclose where the ureters entered the bladder or cloaca. It was finally concluded that the cystoscope, inserted through the lateral fistulous tracts, apparently entered the uterine cavity and not the bladder. X-rays of the gastro-intestinal tract showed a normal stomach and duodenum, with emptying of the former in 6 hours and incomplete filling of the colon in 24 hours. There was a tremendous dilatation of what appeared to be the rectum.

Thirty days after admission to the hospital, a plastic operation, to create an artificial anus, was done. A perineal incision was made in the region where the anus should be and carried down to the pelvic floor, disclosing excellent levator ani muscles and a dimple surrounded by a fairly good sphincter muscle. An artificial anus was created by bringing the rectum through this opening and suturing it so that it protruded outside. Five days later, this protruding loop of the rectum was opened with a coagulating current and a large catheter was inserted. For about two weeks this artificial rectum was irrigated and dilated daily.

Eighteen days later (48 days after admission) an exploratory operation was done, with the object of closing the lateral fistulae. An incision was made from one fistulous opening to the other, allowing the cloacal pouch to be opened wide. Examination revealed a cervix on its upper portion. In the lower anterior midline, at the skin margin, was a fairly normal-looking urethral opening. A sound passed through this entered the bladder, releasing urine. The rectal opening was found next to the skin margin in the posterior mid-line of the cloaca, and was about 2 cm. in diameter. The mucous membrane about the rectal fistula was incised and dissected down to the rectum, and closed by silver wire sutures. A rectal tube was left in the artificial anus. Following this operation, the patient had a normal urethral opening, a vaginal orifice with cervix, and an artificial anus which seemed to have a very good sphincter. It was found that the patient was unable to tell when her bladder was distended, but she was taught to empty it at regular intervals by pressure over the symphysis, and thus was perfectly continent and no longer wet the bed. The wound gradually healed, and the rectum was dilated daily until she was able to be up and about, with control of feces. She was discharged 33 days after the second operation (81 days after ad-

mission), her mother having been taught to keep the rectum dilated by means of a large rectal tube.

Two and a half years later the patient returned for a check-up and excretory pyelograms. The latter showed normal kidney pelves and calyces. They also showed that there were 7 rubber rectal dilators in the dilated upper rectum. The patient's mother said that she knew the dilators had been lost, but did not realize that they had been lost in the patient. The dilators were removed by operation by the general surgical service of the hospital, with no complications. At this time the patient had perfect urinary and rectal control. She was able to empty her bladder without pressure on the abdomen. The results of the plastic operation on the external genitalia were most satisfactory.

IMPOTENCE

A plastic operation for the cure of certain types of impotence has been on trial for almost seven years, and the results in properly selected cases have been so gratifying that it seems worth while to review the essential details of the operation as now performed.

The anesthesia may be either spinal or sacral. The patient is placed on the operating table in an exaggerated lithotomy position, and a No. 19 French sound is passed into the urethra. An incision is made over the bulging part of the perineum, extending in the midline from a point about 10 cm. from the anal margin down toward that structure for about 5 cm. A branch is then made laterally on each side to a point just above the attachment of the crus penis, the completed incision resembling an inverted Y. The incision is deepened through fat and areolar tissue until the corpus spongiosum, surrounded by the bulbocavernosus, and the crus penis (corpus cavernosum) on each side, surrounded by the ischiocavernosus, are exposed. Chromic ribbon gut, studded with an atraumatic needle, is inserted into the lateral edge of the bulbocavernosus muscle, pulled across the belly of the muscle and passed through the other side, with just enough strain to plicate the muscle and produce the exact amount of pressure to reinforce any contraction necessary to aid in producing an erection. Two other similar stitches may be required to tighten the whole muscle. A figure-of-eight ribbon-gut suture is inserted into the ischiocavernosus muscle on each side, care being taken not to injure or unduly compress the fairly numerous nerves and blood vessels in this area. By tightening the suture a reef is taken in the muscle, thus shortening it by approximately 2.5 cm. The wound is closed in layers, without drainage.

An incision 4 cm. long is then made over the suspensory ligament, which is exposed and freed by blunt dissection. A compression suture of ribbon gut is placed in the two leaves of the suspensory ligament of the penis; this, when tied, causes a moderate, persistent compression of the vessels draining the dorsum of the penis, thereby preventing a too-rapid flow of blood out of the organ. This second step, a modification of the original operation, we do because three of the patients in the original series were relieved at first and then became less efficient due to the fact that the dorsal veins of the penis enlarged, allowing the extensive collateral circulation to carry off too freely the stasis necessary for the production and maintenance of erection.

Chart 1 shows the results of this operation to date.

UNDESCENDED TESTIS

Orchidopexy, to be successful, must (1) preserve the blood and nerve supply of the testis,

SUMMARY OF RESULTS OF OPERATIONS FOR RELIEF OF IMPOTENCE TO AND INCLUDING NOVEMBER 1, 1941*

Age Group, Years	Cases Followed: No. of Cases	Per Cent	Average Hospital Days	Results: Successful Number	Successful Per Cent	Improved Number	Improved Per Cent	Unchanged Number	Unchanged Per Cent	Not Followed
30-	19	13	9	14	74	3	16	2	10	2
30-40	43	31	9	37	86	6	14	0	0	1
40-50	45	31	10	26	58	8	18	11	24	7
50-60	29	20	10	15	52	12	41	2	7	11
60+	7	5	12	4	57	2	29	1	14	3
Total	143	100		96	67	31	22	16	11	24

*One hundred and forty-three patients were followed. In addition, there were 24 whom we were unable to follow, making a total of 167 in this recorded series.

Twenty additional patients have been operated upon outside New York, so that we have no record of them.

Chart 1

(2) fix the gland in the bottom of the scrotum without undue traction of the cord, and (3) cure the hernia that almost always accompanies this condition. There are certain cardinal principles involved in reducing an undescended testis to its proper place in the scrotum, namely: (1) each and every blood vessel in the cord must be dissected free from each of its fellows; (2) the fibrous tissue, the real cause of the maldescent, must be excised (the vas is always sufficiently long); (3) the testis must be fixed in the scrotum in such a manner that a cough, sneeze, or other spasmodic contraction will not give a sudden pull on the organ and draw it up to the repaired external inguinal ring before its new position in the scrotum is stabilized.

Many operations for the correction of undescended testes have been devised. In most common use are the operation of Bevan, in which retraction of the testis is prevented by a purse-string suture at the neck of the scrotum, and the procedure of Torek, in which the testis is sutured to the fascia lata of the thigh, from which it is detached, and the scrotum and thigh are separated, at a later operation. The procedure preferred by us is a modification of the Torek operation. For a number of years we have fixed the testis to the skin of the thigh by the simple device of attaching a No. 2 chromic catgut suture to the tunica vaginalis in such a manner as to avoid constricting any vital part of either the testis or epididymis, inserting the suture through the bottom of the scrotum and into the skin of the thigh at an appropriate place, and securing it. The catgut absorbs sufficiently to part in about 8 to 10 days, by which time the testis is firmly fixed in its new location.

ECTOPIC TESTIS

In contradistinction to the undescended testis, which is arrested at some point in the line of normal descent, an ectopic testis is one which moves out of the regular course of descent and actually becomes "lost." These straying organs are sometimes found in the most astonishing places. Cases have been reported in which the testis has gravitated to the perineum, the penoscrotal junction, Scarpa's triangle, or the anterior-superior spine of the ilium on the superficial surface of the aponeurosis of the external oblique muscle. Occasionally a testis may descend through the opposite inguinal canal, and rarely one will descend into the true pelvis, as in two cases seen by us.

It is our opinion that ectopic testis is an operable condition. The testis may be successfully reduced in a viable condition to its proper place in the scrotum and fixed to the skin of the thigh by means of chromic catgut anchor stitches.

In one of the cases of ectopia testis pelvicis seen by us, a very interesting case reported by Robert W. Hunt from St. Clare's Hospital, both testes were found to be attached to the sigmoid, low down, and were successfully reduced to their proper places in the scrotum.

The other case, reported by Lowsley and Curtis, occurred in Bermuda. A brief report of this follows:

> The patient, a colored man, had presented himself to Dr. E. A. Cann, of Somerset, Bermuda, four years earlier suffering from an acute neisserian infection of the urethra. During the course of this he developed prostatitis and bilateral epididymitis, with pain in the region of the rectum and acute pain in the mid-line of the abdomen. This subsided promptly, but the prostatitis recurred on two subsequent occasions. During the last attack, sulfanilamide was used, with prompt relief. The patient was normal sexually.
>
> Physical examination showed no abnormalities except for the external genitalia. No testes could be palpated either in the scrotum or in the inguinal canal. There were no hernias.
>
> Under ether anesthesia, a 10 cm. incision was made obliquely upward and outward from the spine of the pubis over the left inguinal ring. The fascia was incised and the incision deepened into the inguinal canal. The bladder was inadvertently incised, but was immediately repaired and drained. The peritoneum was opened, and after considerable search a testis was located in the left iliac fossa outside of the iliac vessels. The testis was mobilized, it being necessary to tie off several aberrant vessels in the process. The vas was of ample length. The main mass of vessels forming the cord was held in a tight fibrous envelope. This was excised, after which the vessels were found to be sufficiently long to permit the testis to be brought into the left scrotum, where it was fixed with a double chromic catgut suture passed through the scrotal sac and the skin of the thigh. The peritoneum was closed with plain catgut No. 2; the inguinal canal with interrupted chromic ribbon gut sutures; the fascia with cromic catgut No. 2, and the skin with metal clips.
>
> On the right side, a similar 10 cm. incision was made and deepened through the fascia, later being extended upward for 3 cm. The floor of the inguinal canal was opened and the peritoneum incised. The vessels of the cord were visualized, but ended blindly at the internal ring of the inguinal canal. The patient was then placed in the Trendelenburg position and the intestines packed off. Only by following the vas deferens from its terminus in the ampulla was the testis finally discovered in its retroperitoneal position at the level of the right kidney hilus, where it developed in embryo. In other words, the testis had not descended at all. The posterior layer of the peritoneum was incised and the testis and vas isolated. Unfortunately, the regular blood supply was entirely lacking, ending blindly at the internal inguinal ring. The testis was entirely supplied by aberrant vessels, which had to be tied off, leaving the artery and veins of the vas, which Bevan has repeatedly stated

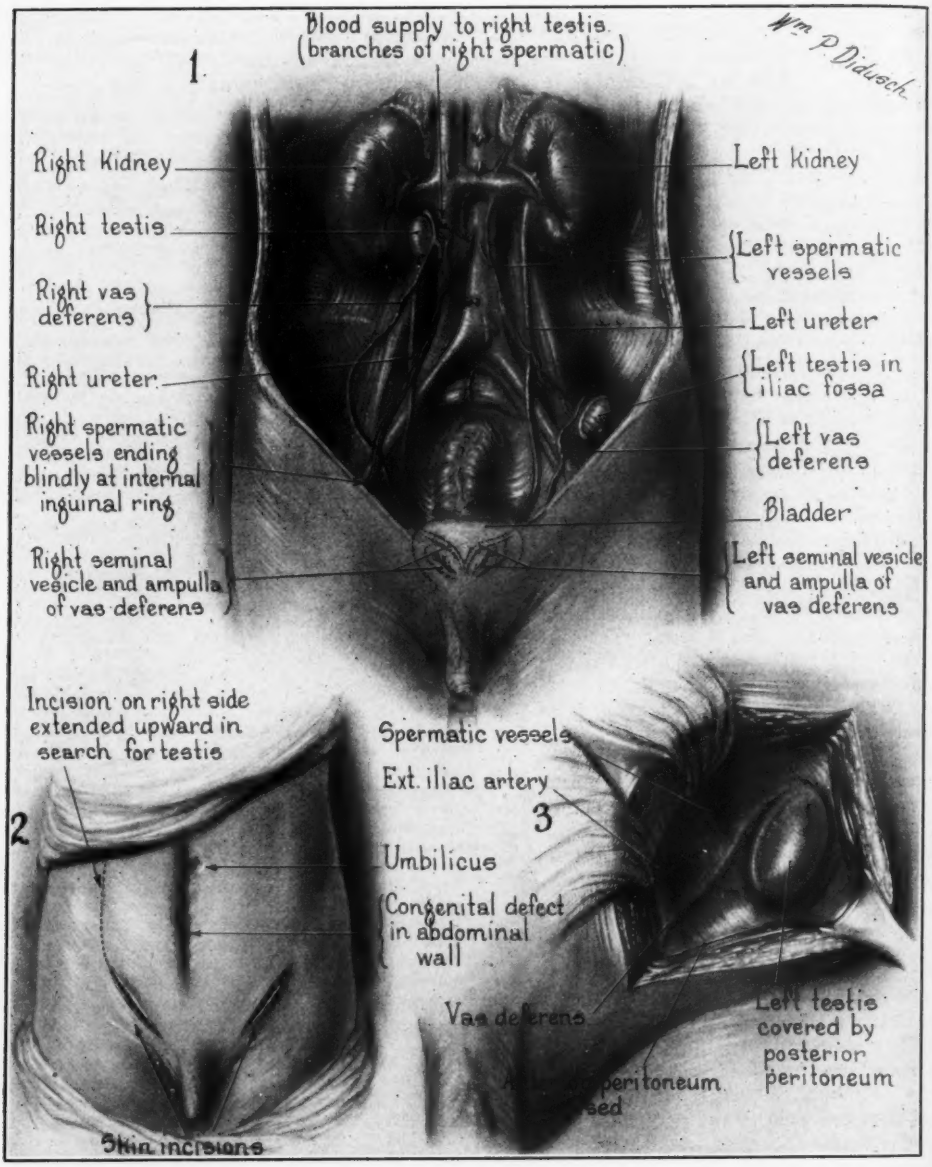

Fig. 1

Unusual case of bilateral ectopic testes. (1) Condition present. Right testis in region of hilus of right kidney. Its blood supply is from the spermatics by way of small branches; the main trunks of the spermatics end blindly in region of internal inguinal ring. Left testis in region of iliac fossa. (2) Skin incisions to expose testes; right incision was extended upward. (3) Shows left testis exposed, and found to be in iliac fossa, covered by posterior peritoneum.

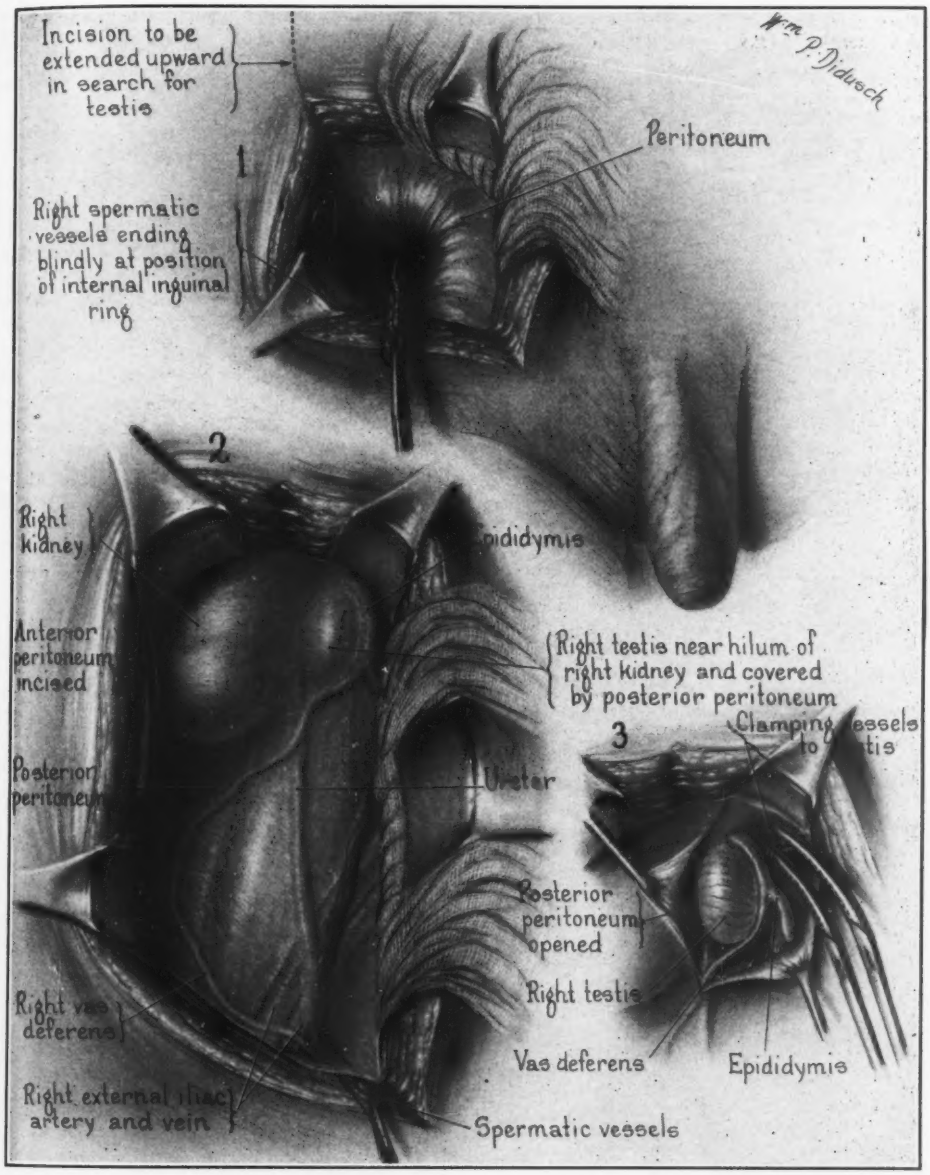

Fig. 2

Bilateral ectopic testes. (Right side) (1) Shows spermatic vessels ending blindly in region of internal inguinal ring, just under peritoneum. (2) Incision extended upward toward kidney region. Right testis found near hilus of kidney, covered by posterior peritoneum. Right vas deferens, ureter, and iliac vessels could be seen. (3) Posterior peritoneum incised; right testis exposed and blood vessels, branches of the right spermatics clamped, divided and ligated. Note unusual shape of epididymis.

are sufficient to maintain the organ. The testis was reduced without difficulty to its proper place in the scrotum, where it was fixed to the thigh in the manner already described. The posterior and anterior layers of the peritoneum were closed with No. 2 catgut; the muscle with interrupted chromic catgut No. 2; the floor of the inguinal canal with chromic ribbon gut; the fascia with chromic catgut No. 2, and the skin with metal clips (*Figs. 1, 2* and *3*).

Unfortunately, this patient had a very stormy postoperative course and finally expired from myocardial failure.

THE URETHRA

Incontinence of Urine

Incontinence of urine is a fairly common complaint in both sexes, but is much more frequent in women. However, it is considerably more common in men than it formerly was, due to the fact that more men are now having operations for the removal of the prostate gland than ever before in the history of surgery. This unfortunate condition plays no favorites, and may follow transurethral, perineal or suprapubic prostatectomy, in the order mentioned. In women, it usually follows protracted or difficult labor; but it is not unusual to elicit the history that a young woman or girl has always had a "weak" bladder, losing a few drops or more of urine upon exertion.

In men, the cure of urinary incontinence is accomplished by plication of the bulbocavernosus muscle with chromic ribbon gut. This material is necessarily used because it does not tear through the delicate musculature and, being an animal membrane, it allows the new fibroblasts to grow into it, thus establishing a firm scar which does not stretch, but also does not stricture the urethra because it does not completely surround it.

When incontinence of feces accompanies the urinary incontinence, as often happens in spina bifida, cure of the former may also be accomplished by plicating the rectum by means of mattress sutures of chromic ribbon gut as described by us in the *Journal of Urology* in February, 1939 (*Fig. 4*).

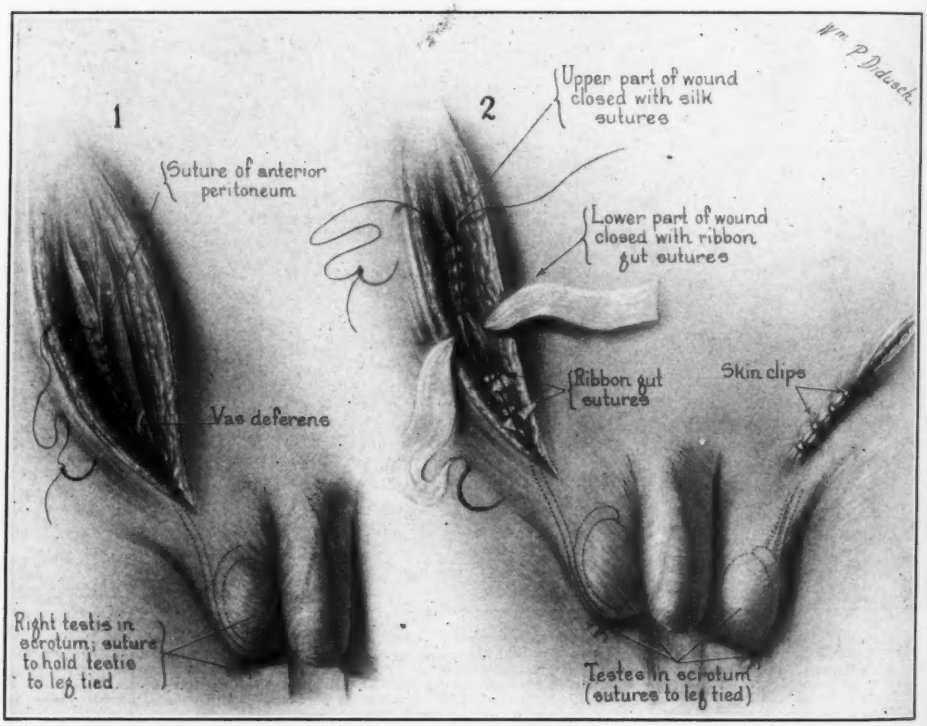
Wm P Didusch
1
2
Upper part of wound closed with silk sutures
Suture of anterior peritoneum
Lower part of wound closed with ribbon gut sutures
Ribbon gut sutures
Skin clips
Vas deferens
Right testis in scrotum; suture to hold testis to leg tied.
Testes in scrotum (sutures to leg tied)

Fig. 3

Bilateral ectopic testes. (1) Right testis in scrotum; suture to leg tied. Closure of anterior peritoneum. (2) Showing both testes in the scrotum. Closure of lower part of wound with ribbon gut, upper part with silk sutures. Skin closed with skin clips.

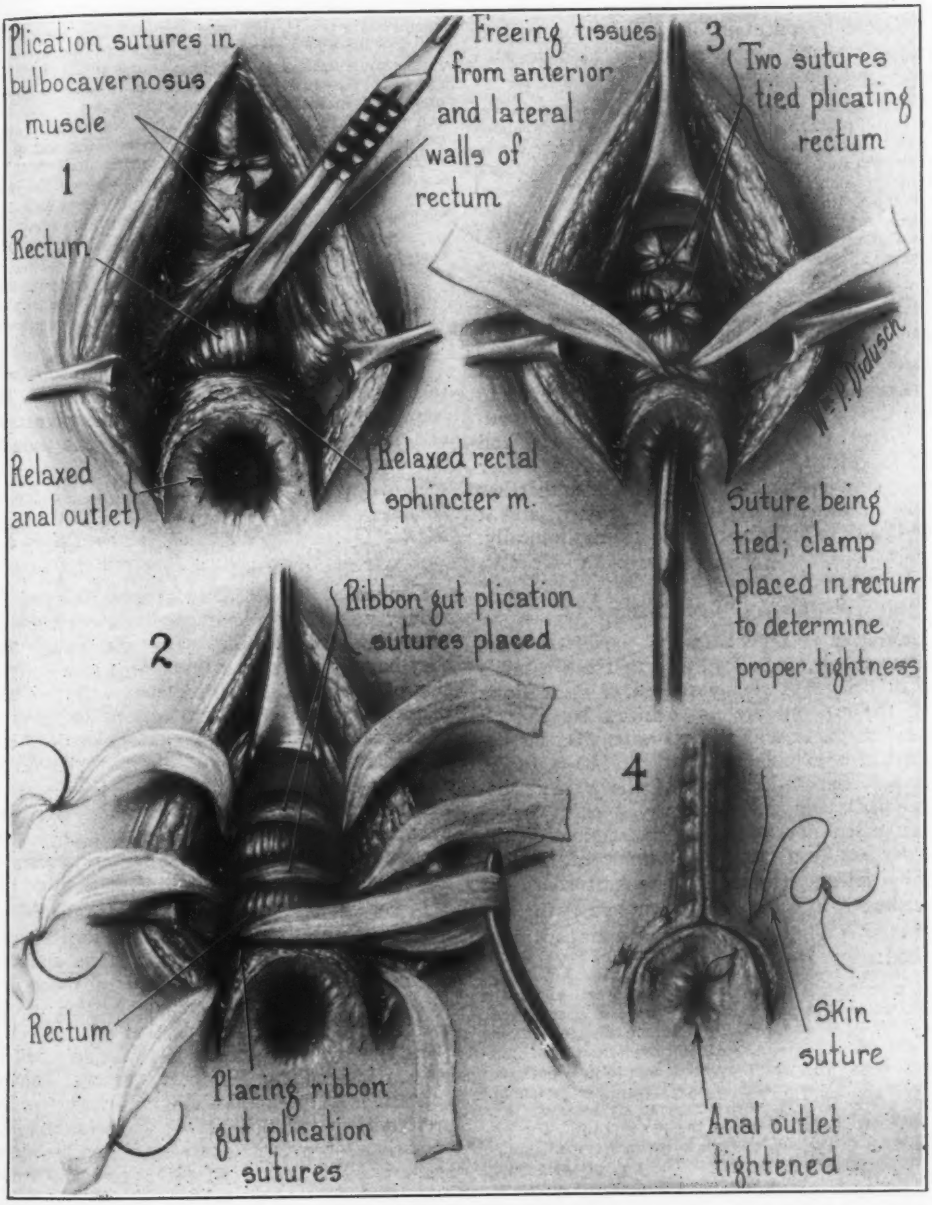

Fig. 4

Lowsley-Hunt plication operation for incontinence of urine and feces in the male. (1) The bulbocavernosus muscle has been plicated for cure of urinary incontinence. Freeing anterior and lateral walls of the rectum from surrounding tissues. (2) Placing chromic ribbon-gut plication sutures partially around the rectum. (3) Plication suture being tied; clamp placed in rectum to determine proper tightness. (4) Final skin closure.

In the female, the cure of urinary incontinence is effected by plication of the anterior wall of the urethra and the neck of the bladder with ribbon gut, as follows:

Under spinal anesthesia, the patient is put in the lithotomy position. A curved incision is made above the urethra and deepened in such a way that the top of the urethra and neck of the bladder are exposed. Using chromic ribbon gut, the neck of the bladder is plicated over a No. 10 French catheter by means of a mattress suture. Additional sutures of the same material are placed along the entire urethra so that it is constricted rather tightly about the small catheter extending through it. The subcutaneous tissue is approximated with plain catgut and the skin closed with silk. A small wick drain is left in place for 24 hours, and the catheter is fixed in position by means of a silk suture.

After any of these operations, the patient has to be given lessons in voiding. It is our custom to have the postoperative patient empty the bladder every hour (by pressure, if necessary) and then lengthen the interval until finally a 4-hour period is easily maintained. A bladder that has been constantly empty must be trained, and it soon begins to function more or less rhythmically.

THE BLADDER

Exstrophy of the Bladder

Exstrophy of the bladder is one of the most distressing physical conditions that can assail a human being. A steady stream of urine flows from the exposed ureteral orifices, bathing all dependent parts, and no apparatus thus far devised has been able to handle it satisfactorily. The red, tomato-like wall of the bladder becomes irritated, and in untreated cases malignancy often develops. Reconstruction of the bladder has been frequently attempted, but has usually failed. Ordinarily, the best course of procedure is to transplant the ureters to the sigmoid, preferably by the following extraperitoneal method:

An incision is made from a point 5 cm. above the umbilicus, in the nipple line, down to a point 5 cm. below the crest of the ilium and then directed toward the mid-line. This is deepened through the fascia, muscle and deep fascia, and the peritoneum pushed back and retracted until the ureter is identified. The lower end of the ureter is clamped and severed on a bevel. A 3 cm. incision is then made in the peritoneum. The sigmoid is identified and a small knuckle brought through the incision in the peritoneum. The wall of the sigmoid is then attached by a chromic No. 0 catgut suture on an atraumatic needle to the entire edge of the peritoneal incision, thus extraperitonealizing the wall of the gut into which the end of the ureter is to be transplanted.

An incision is next made through the muscular wall to, but not into, the submucosa. The ureter is buried in the groove thus made, about 2 cm. of its end extending beyond the new bed. The muscularis of the sigmoid is sutured over the ureteral wall, which is fixed at one or more points, the ureter traveling thus through the gut wall for 4 or 5 cm. At the lower end of the incision in the muscularis a purse-string suture is arranged, but not tied. A suture is then tied to the beveled end of the ureter; an incision is made into the lumen of the gut; and the suture is passed through it and brought out 2 cm. below the incision into the lumen. The end of the ureter is pulled through and the suture made fast, fixing the lower end of the ureter to the wall of the intestine. The purse-string suture is then pulled tight and tied, and the entire area covered with sulfanilamide powder. The ureter is thereby transplanted extraperitoneally, with a minimum of exposure to soiling.

Two or three weeks later the opposite ureter is similarly treated and a month thereafter the exstrophied bladder is excised and the raw areas are covered with skin.

With increased skill in technic our results with this procedure have improved greatly. The combined use of extraperitoneal transplantation and sulfanilamide applied locally has reduced the mortality to almost nil.

THE KIDNEY

Plastic Operations On the Kidney Pelvis and Ureteropelvic Junction

Congenital and acquired abnormalities of the kidney pelvis and ureteropelvic junction are almost infinite in variety. The one feature that they have in common is that they cause obstruction of the ureteropelvic junction, with resultant dilatation of the pelvis and calyces and gradual destruction of the kidney parenchyma. When the obstruction is caused by aberrant vessels, the situation is usually easily corrected by severing the offending vessels after ligature. If, however, the condition is due to a stricture at the ureteropelvic junction, a more serious problem is presented.

Many different types of operations for the relief of such obstructions have been devised, evidence that the problem is a serious one, and that no one method is the standard of procedure. Chief among them are: reimplantation of the ureter in the dependent portion of the pelvis (first employed by Kuester in 1891 and modified by von Lichtenberg, Wildbolz, Quinby, Walters, and others); the application to stricture at the ureteropelvic junction of the Heineke-Mikulicz operation for pyloric stenosis (Fenger); lateral anastomosis between the pelvis and the ureter (von Lichtenberg, Walters); enlarging the ureteropelvic outlet by making a Y-shaped incision on the pelvis and down the ureter, across the narrow portion, and closing the Y as a V (Schwyzer; modified by Foley); the Rammstedt pyloroplasty operation applied to the ureter (D. M.

Davis and others); and the operations of Young and Hellström for aberrant vessel, employing resection of part of the pelvis with closure in such a manner as to draw the offending vessels into a position where they will not obstruct the ureter.

Important factors in the success of plastic operations on the renal pelvis are (1) the eradication or minimizing of any existing kidney infection before operation is attempted, infection being the cause of most failures; (2) the establishment of surgical drainage, usually by nephrostomy. The sulfonamide drugs, used in the wound as well as orally, have been most helpful in reducing infection in these cases.

Removal of Segment of Double Kidney with Aberrant Ureter

An aberrant ureter always arises distally to the normal ureter and with rare exceptions leads to the cranial segment of a double kidney. This segment is usually sac-like or a poorly functioning remnant with rudimentary cortex. Almost always it is infected. The ureter itself is generally dilated and tortuous. In the female, the ectopic orifice is usually located in the urethra or vestibule, but has been found in the vagina or uterus; in the male, it is found most often in the prostatic urethra or seminal vesicle. There is a predominance of reported cases in females over males of nearly 2 to 1; but as this anomaly is usually asymptomatic in males, and rarely recognized clinically, this sex variation is not significant. In females, there is a constant seepage of urine, which usually is purulent. Since the segment of kidney served by the aberrant ureter is almost always functionless, or nearly so, the operation of choice is resection of the accessory organ, which usually is buried in the upper pole of the normal kidney on that side.

A typical case of this type, and its operative cure, reported by Lowsley and Conroy,* follows:

F. P., a 23-year-old female, had had slight but constant leakage of urine since birth, necessitating the wearing of a pad. She had been examined in childhood and told that nothing could be done until adult life. There had been no change since except that the urine had become more purulent. She had always urinated normally and passed normal amounts, and there were no other complaints. One year before her admission to the Brady Foundation, she had been examined and a diagnosis of vesicovaginal fistula made. It was for the operative repair of this that she was referred to the author (O.S.L.). Preliminary examination, however, failed to reveal a fistula and she was admitted to the hospital for further study.

The general physical and pelvic examinations showed nothing abnormal except for a constant seepage of thin, purulent fluid, evidently urine, from the vestibule. Cystoscopy and retrograde pyelography were done without difficulty. A normal bladder, with normally placed ureteral orifices, was found. More careful examination of the vestibule then disclosed that the discharge came from a point on the urethral floor just without the meatus. A urethral catheter was inserted and a retrograde pyelogram made. This showed the catheter curled up in the markedly dilated distal end of the aberrant ureter. The entire ureter filled well. It was seen to be moderately dilated throughout its length, and extended to a point above the pelvis of the left kidney, where a sac-like secondary pelvis was visualized. A diagnosis of double kidney with an aplastic segment drained by the ectopic ureter was made, and operation advised.

Under spinal anesthesia, the left kidney was exposed through the usual lumbar incision. The accessory kidney segment was found to form the upper pole of a double kidney and to be enclosed within the same capsule. The kidney was mobilized. The supernumerary ureter was posterior and somewhat lateral to the normal ureter. It was clamped and severed low down and the stump ligated and cauterized. The kidney capsule was then incised, exposing a shallow groove marking the division between the two kidney segments. The cranial segment received its arterial supply through a branch from the main renal artery. This was clamped, after which the cranial segment (about one-sixth of the renal mass) assumed a slightly dusky color. The venous return was evidently through the normal kidney segment. By sharp dissection through the line of demarcation, the accessory kidney was excised. Very little bleeding ensued. A pad of fat was placed on this fossa and the normal kidney margins approximated by interrupted sutures of chromic ribbon gut inserted through the capsule.

Postoperatively there was moderate infection of the wound, but the patient was discharged from the hospital on the twenty-third day, with the wound well healed and with her incontinence cured.

*To be published.

HEMINEPHRECTOMY

Heminephrectomy is indicated in many kidney lesions in addition to the type just described. Among the more common are cysts, localized non-tuberculosis abscesses, and calculous pyonephrosis limited to one pole. We are of the opinion that it is never advisable to do a heminephrectomy in a case of renal tuberculosis, no matter how localized the lesion may seem to be, because in every case that has come to our attention total nephrectomy has been the ultimate outcome.

The operation we prefer is the one described by us before this body in 1933. This is done as follows:

Under spinal anesthesia, the kidney is exposed and mobilized in the usual manner. The portion of the kidney to be removed is outlined and a V-shaped incision is made in such a manner that the diseased portion of

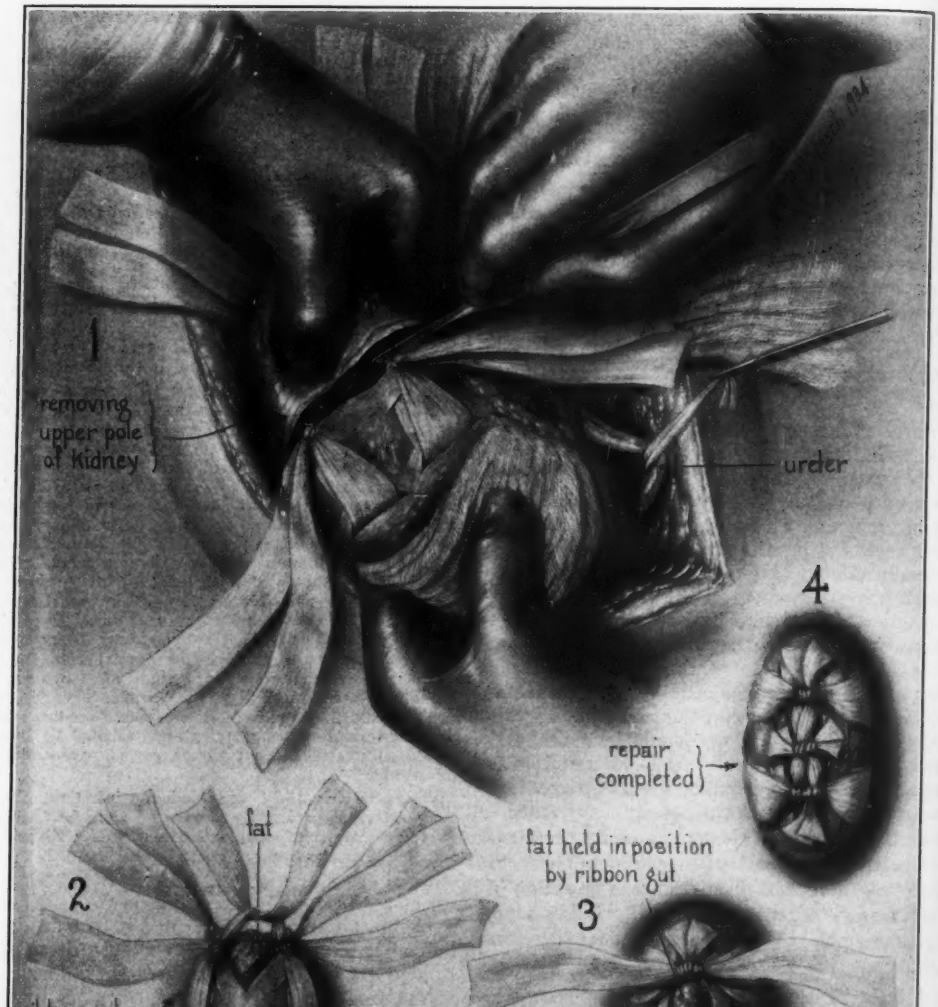

Fig. 5

Heminephrectomy, using ribbon gut for repair of kidney. (1) The kidney has been delivered, and the ribbon gut placed in position on the anterior and posterior surfaces, being drawn through the kidney capsule. Resecting upper pole of kidney. (2) Lateral view, showing ribbon gut being tied over pad of fat applied to cut surface to control bleeding. (3) Kidney viewed from upper pole, with hemorrhage controlled. (4) Loose ends of ribbon gut are tied together.

the organ is excised. A pat of fat is buttered over the cut surface of the kidney and the edges of the wound drawn together over it by means of ribbon gut sutures properly placed through the kidney capsule, a tube drain being placed in the renal pelvis. The kidney is replaced in the renal fossa, and the wound closed in the usual manner, with drainage (*Fig. 5*).

CONCLUSION

In conclusion, may it be said that plastic surgery upon organs of the genital and urinary tracts offers a fascinating opportunity for meticulous surgical technic. No greater thrill can come to the surgeon than that afforded by curative procedures on those pitiful patients suffering from deformities of the external genitalia and bladder who are often ostracized from the ordinary pursuits and pleasures of the normal human being.

PAROXYSMAL TACHYCARDIA OF SUPRAVENTRICULAR ORIGIN*

MANAGEMENT OF THE ACUTE SEIZURE

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and

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Paroxysmal rapid heart action always presents an intriguing therapeutic problem and particularly is this true in the case of paroxysmal tachycardia of supraventricular origin which constitutes about one-fourth of the cases encountered. The types included in this discussion are the two ectopic varieties, namely: the auricular and auriculo-ventricular nodal, and the rare nomotopic variety, paroxysmal sino-auricular tachycardia. Clinical differentiation of the three types is impossible and is entirely dependent upon electrocardiographic study. Fortunately, however, the same therapeutic measures are applicable to each.

In many instances the attacks may be of such short duration and so innocuous as to require no therapy. However, in the majority of cases the indications are to bring about a cessation of the attack as early as possible in order to relieve the distressing and disturbing symptoms which accompany them and to obviate the possibility of complications, which although rare, do occur.

The available therapeutic measures may be conveniently discussed under three headings: physical measures, drug therapy, and surgery. Generally speaking, the physical measures should be tried first and drug therapy resorted to when the former is ineffectual. Surgical therapy should be contemplated only in those very rare instances which fail to respond to all other measures.

PHYSICAL MEASURES

The various physical measures listed in *Table 1* are often successful in bringing about prompt cessation of the attack. In each instance the modus operandi is stimulation of the vagus nerve. In view of the fact that repeated attacks are the rule it is highly beneficial and reassuring to the patient if he knows of something he himself can do which will terminate the seizure. Consequently all of the simple procedures should be outlined for the patient in detail so that they may be tried in succession. Quite often a patient will find a certain measure which will consistently give the desired result.

Because of their simplicity and high degree of efficacy, Valsalva's experiment (an attempt to expire forcibly with the glottis closed after a deep inspiration) or Muller's experiment (an attempt to inspire forcibly with the glottis closed after deep expiration) should be tried first. The former is more often successful. The procedure should be described in detail to the patient and emphasis should be placed upon continuing the experiment as long as the breath can be held. If unsuccessful at first, the maneuver should be repeated as rapidly as possible for three or four times.

Vomiting is often successful in terminating a seizure, particularly in those cases in which overloading of the stomach and gastric distension are definite precipitating factors. Emesis

PAROXYSMAL TACHYCARDIA OF SUPRAVENTRICULAR ORIGIN: MANAGEMENT OF THE ACUTE SEIZURE

PHYSICAL MEASURES

(1) Induction of emesis	(6) Olfactory stimulation
(2) Valsalva's experiment	(7) Ocular pressure
(3) Muller's experiment	(8) Carotid sinus stimulation
(4) Calisthenic motion or changes in posture	(9) Application of a tight abdominal binder
(5) Ingestion of cold solutions	

Table 1

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*Read in Section on Medicine, Southern Medical Association, Thirty-Fifth Annual Meeting, St. Louis, Missouri, November 10-13, 1941.

*From the Cardiovascular Service of the John Sealy Hospital, Department of Practice of Medicine, Medical Branch, University of Texas.

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may be induced in one of several ways: mechanical irritation of the pharynx; the ingestion of hot solutions of sodium bicarbonate; sodium chloride; or the use of emetics such as syrup of ipecac or apomorphine, preferably the former. Changes in posture, such as lowering the head, flexion or extension of the trunk, forcible extension of the arms, or other calisthenic exercises occasionally are successful. During such maneuvers the breath is usually unconsciously held, and this may be the source of vagal stimulation. The rapid drinking of very cold water has been reported as terminating an attack. Olfactory stimulation with smelling salts or aromatic spirits of ammonia rarely produces the desired effect. The application of a tight abdominal binder has been used successfully by some patients. The placing of an ice bag over the precordium is often recommended, but its efficacy is somewhat doubtful.

If the simpler measures have failed, more effective methods of mechanical stimulation of the vagus should be tried. Pressure over the eyeballs bringing into play the oculo-cardiac reflex is routinely recommended, but in our experience fails in most instances. Furthermore, the procedure usually causes considerable pain. Stimulation of the carotid sinus is the most effective mechanical method of producing powerful vagal stimulation. White[1] states that success will be obtained by this method of vagus stimulation in only about 10 per cent of the cases. Our experience would indicate that the procedure is successful in approximately 20 to 30 per cent of all cases, whereas in the elderly subject this figure will be more than doubled.

Factors which govern whether or not carotid sinus stimulation will produce the desired result are: the age of the patient, the presence of hypertension and arteriosclerosis, and the technic employed. The sensitivity of the carotid sinus reflex increases with age. The existence of arteriosclerosis and hypertension likewise appear to increase the sensitivity of the reflex. The third factor, the technic employed, is, in our opinion, very important. The sinus itself must be actually stimulated. Pressure applied promiscuously in the region of the carotid sheath will not be effective. The application of a standardized procedure cannot be too strongly recommended. Because of the fact that syncope occasionally occurs the patient should be placed in the recumbent position. In view of the predominant effect of the right vagus on the auricular musculature the right carotid sinus should be stimulated first. With the examiner standing by the right side of the patient, the palm of the left hand should be passed behind the patient's neck with the head slightly elevated and turned toward the examiner in order to relax the sternomastoid muscle. The upper border of the thyroid cartilage should then be located by the ball of the thumb and gentle pressure exerted between the former structure and the sternomastoid muscle. By moving the thumb up and down along the carotid artery in that location the bulbous dilatation and pulsation of the carotid sinus can usually be felt. Pressure should then be gradually increased impinging the carotid sinus between the thumb and the vertebral column. If sudden pressure is applied, the artery may slip from underneath the finger. As pressure is applied the stethoscope should be held over the precordium and at the first sign of slowing, stimulation should be stopped. In this way the occasional resulting syncope with or without convulsions will be reduced to a minimum.

If several trials are unsuccessful on the right carotid sinus, the left should then be stimulated. In those instances of junctional type, left carotid sinus stimulation would appear more likely to be effective in view of the fact that the predominant effect of the left vagus is exerted on the junctional tissues.

DRUG THERAPY

When the physical measures are ineffective, drug therapy must be resorted to. In *Table 2* are listed those drugs which have been used successfully in the management of an acute seizure and which appear to have specificity and a sound physiologic as well as a pharmacologic basis.

In view of the morbid physiology involved, drugs with vagomimetic properties would seem to offer the greatest promise of success. Acetyl-beta-methylcholine chloride ("mecholyl"), "prostigmine" methyl sulfate, and possibly physostigmine salicylate are members of this group. The initial report on the effectiveness of mech-

PAROXYSMAL TACHYCARDIA OF SUPRAVENTRICULAR ORIGIN: MANAGEMENT OF THE ACUTE SEIZURE

DRUG THERAPY

(1) "Mecholyl"	(7) Sedatives
(2) "Prostigmine"	(8) Opiates
(3) Calcium salts	(9) Ergot derivatives
(4) Cinchona alkaloids	(10) Physostigmine
(5) Cardiac glucosides	(11) Parathyroid hormone
(6) Magnesium sulphate	

Table 2

olyl in paroxysmal tachycardia was made by Starr.[2] His results have subsequently been confirmed by other investigators. It is recommended that the drug be given subcutaneously in doses of 5 to 50 mg., starting with the small dose. If cessation of the paroxysm does not occur concomitant with the appearance of the other systemic symptoms induced by the drug, namely: a feeling of warmth about the head and neck, salivation, lachrymation, and sweating, the site of the injection should be vigorously massaged to hasten absorption. The failure of systemic symptoms to appear is an indication that the initial dose has been too small. If, after massage, the desired effect is still not obtained, carotid sinus stimulation should be instituted. This maneuver is usually successful, although it had been without effect prior to the administration of the drug. The effect of mecholyl is transitory, lasting only a few minutes, and if it is felt that the initial dose was inadequate, a larger second dose may be given safely after ten minutes have elapsed. We have used acetyl-beta-methylcholine chloride in eleven cases of paroxysmal tachycardia. In seven instances the treatment was promptly successful. In a case of nodal origin, the rate of impulse formation was slowed from 160 to 120 per minute, but the ectopic focus persisted. Spontaneous restoration of sinus rhythm occurred some thirty minutes later. In the remaining three cases the drug was ineffectual. When the optimal dosage is carefully approached, toxic effects are minimal and, in our experience, have not been of any consequence. However, should they occur, atropinization causes a prompt disappearance of all symptoms.

Fig. 1 (top) and Fig. 2 (bottom)
The technic of carotid sinus stimulation.

The cholinergic properties of "prostigmine" prompted Waldman and Moskowitz[3] to study its effect on the normal heart and on paroxysmal auricular tachycardia. In one case of the latter disorder the drug was given during four separate attacks with prompt control in an average of five and one-half minutes after injection. Electrocardiographic studies revealed that before restoration of a normal sinus mechanism, varying grades of auriculo-ventricular block occurred. Pelner[4] treated six attacks of paroxysmal tachycardia in four patients with "prostigmine." Two seizures were stopped twelve and eighteen minutes, respectively, after injection of the drug. In the remainder, carotid sinus stimulation was successfully used fifteen minutes after "prostigmine" was given, whereas before it was ineffective. No electrocardiographic studies were carried out in these cases.

The action of "prostigmine" is apparently dependent upon its effect on the catabolism of acetylcholine which is thought to be the chemical intermediary between the parasympathetic nerve impulse and the response in the effector tissue. Acetylcholine when liberated is hydrolyzed probably by an enzyme cholinesterase into acetic acid and choline, the latter substance possessing only to a very slight degree the properties of its precursor. Experimentally it has been shown that the effect of "mecholyl" can be enhanced and prolonged by the administration of "prostigmine." This is presumably due to the fact that "prostigmine" inhibits or slows the hydrolyzation process, thereby complimenting its effect. The dose employed is one or two mg. given subcutaneously or intramuscularly, preferably the latter. Our experience is limited to three cases. In one case the seizure terminated five minutes after the injection of 2 mg. of the drug. Just as in the case of Waldman and Moskowitz, high-grade auriculo-ventricular block of varying grades was observed. In the second case the attack stopped five minutes after the administration of 1 mg. of "prostigmine," but promptly recurred and was subsequently controlled by quinidine. In the third case the drug was ineffectual. In view of the synergism that exists between "mecholyl" and "prostigmine," the combined use of the two drugs in small doses is suggested and will be cautiously tried.

The effectiveness of the cardiac glucosides, particularly digitalis, in the treatment of the immediate attack as well as in preventing recurrences has been demonstrated by many investigators. Full digitalization by mouth in the usual manner may be carried out. However, in the severe intractable cases associated with signs and symptoms of impending or actual heart failure, the parenteral route should be the method of choice. Under such circumstances the chemically pure, crystalline, stable glucosides derived from digitalis lanata, "lantoside C" or "digoxin," would appear to have many advantages over the available parenteral preparations of digitalis purpurea. Fahr and LaDue[5] reported five cases which were promptly controlled by the intravenous administration of 8 c. c. of "lantoside C," normal sinus rhythm being established within five minutes in two cases, within ten minutes in another, and within two hours in two more. In a case of paroxysmal auricular tachycardia complicating cardiac infarction, the drug was ineffective. Junet[6] reported seven cases treated similarly with "lantoside C" without a failure. We have used the cardiac glucosides in seven cases with one failure. In three instances the digitalis lanata glucosides were employed and in the remaining three parenteral digitalis purpurea preparations. Other cardiac glucosides such as strophanthin and oubain have been reported as being likewise effective.

Wolffe and Bellet[7] advocated the use of calcium salts intravenously in the management of the acute seizure. Three out of five cases responded promptly to the intravenous administration of 10 to 20 c. c. of 10 per cent calcium gluconate. The pharmacologic basis for their action is the depressant effect of the calcium ion on the irritability of the myocardium. Magnesium sulfate[8] has also been reported as being efficacious in terminating an attack. The dose employed was 15 to 20 c. c. of a 20 per cent solution.

Quinine and quinidine therapy have been used for many years in the management of paroxysmal tachycardia and these drugs are undoubtedly efficacious in many instances.[1] It is recommended by some that they be given intravenously; however, the consensus of opinion appears to be that this should be done only as a last resort in view of the inherent dangers incident to the parenteral administration of any of the cinchona alkaloids. Sudden death has been reported from their use in the treatment of malaria. The oral route is, therefore, the method of choice. Therapeutic failures are often the result of inadequate dosage. A test dose of 3 to 5 grains of quinine should be given to determine the existence of sensitivity to the drug. If no toxic effects are noted, five grains every hour for five to six doses should be given or until toxic manifestations such as nausea or vomiting appear.

Physostigmine,[9] ergotamine tartrate, and parathyroid extract[10] have been reported as successful in stopping attacks. We have had no experience with any of these, and they represent isolated reports without confirmatory studies by others.

No discussion of the drug therapy of paroxysmal tachycardia would be complete without reference to the value of sedation. In most instances, particularly in the early attacks, extreme anxiety and apprehension are prominent features of the clinical picture. Bromides, barbiturates, or opiates may be used and are often very helpful. Morphine exerts no specific effect; however, the emesis which it frequently induces may terminate an attack.

In view of the number of therapeutic agents available, the question immediately arises as to which drug should be chosen. This should depend upon a consideration of several factors:

The existence of organic heart disease and if present, the type and degree;

The urgency or seriousness of the case as determined by symptomatology and physical findings;

The circumstances surrounding an attack as during a surgical operation or labor;

Known contraindications to the drugs; and

Toxicity of the drugs.

Generally speaking the simpler and less toxic drugs should be tried first, the more heroic measures to be reserved for the seriously ill patient. "Mecholyl," "prostigmine," and calcium salts, provided no definite contraindications exist, may be safely used in the majority of patients and probably should be tried in that order.

In the presence of organic heart disease, particularly if advanced and associated with anginal or congestive heart failure or symptoms of impending decompensation, the use of the cardiac glucosides would appear to receive the first consideration. Caution, however, should be exercised in the advanced cases of coronary disease, particularly if cardiac infarction has occurred recently. When no organic heart disease exists and "mecholyl" or "prostigmine" is ineffective, quinidine by mouth would be the drug of choice. "Mecholyl" is contraindicated in patients with asthma, extensive bronchopulmonary disease, and advanced coronary artery disease. The use of the calcium salts should be studiously avoided in the digitalized patient. Sensitivity to the cinchona alkaloids should be determined. Because of the toxicity of quinidine it is questionable in our minds whether the drug should ever be given intravenously.

SURGICAL THERAPY

Occasionally cases of paroxysmal tachycardia are encountered which are refractory to all other methods of treatment and in which the attacks occur so frequently and are of such long duration as to render the patient an invalid. In this type of case it seems justifiable to recommend certain surgical procedures. In 1924, Leriche[11] suggested removal of the stellate ganglion in such cases. The physiologic basis for such an operation is that sympathetic stimulation is abolished in as much as the accelerator nerves to the heart pass through this structure. This end may be accomplished temporarily by injection of the ganglion or the surrounding tissues with an anesthetic, or perhaps permanently by using alcohol, or by actual removal of the ganglion. Up to the present time approximately eleven cases[12] have been reported using one or another of the above named procedures. In some instances the results have been spectacular, the seizure being terminated almost immediately. In others the results were not so dramatic, but the frequency and duration of the seizures appeared to be definitely decreased. It would seem logical to first determine the effect of temporary interruption of the nervous pathways with a local anesthetic. If a satisfactory result is obtained, injection with alcohol or removal should then be considered.

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DISCUSSION (*Abstract*)

Dr. Drew Luten, St. Louis, Mo.—Without recourse to records, it has been my impression that paroxysms of supraventricular origin are encountered more commonly than those originating in the ventricle, if one includes those instances in which the attacks are of relatively brief duration. In many such cases the patient has already discovered some procedure which commonly terminates the paroxysm.

If this and other physical measures enumerated by the author fail, after reassuring the patient it would appear to be good therapy to administer appropriate sedatives and expect cessation within a reasonable time before ordering a specific drug. I am glad that the author emphasizes the desirability of acquainting the patient with various procedures that may be effective. One that frequently is useful is a modified Valsalva in which the patient inspires deeply, and then without exhaling, bends forward. It combines change of position with other favorable factors.

A word of caution might be appropriate with regard to the rapid drinking of very cold water, particularly in older patients. Several cases of myocardial infarction so precipitated have come under my observation.

In cases of anginal failure there might be some question of the propriety of giving digitalis preparations just as the author reminds us there is in acute myocardial infarction, but certainly in congestive failure the glucosides would appear to be ideal.

If we exercise the care in the use of mecholyl that Dr. Schwab observed, and if we follow his advice with reference to the choice of drug appropriate to the case at hand, I am sure that our results will be much more favorable in these annoying and often distressing cases.

Dr. J. E. Hirsh, Birmingham, Ala.—I recall a patient whom I treated for ten years prior to her death. She had been having these attacks on the average of once every four to six weeks since she was 21 years of age. She finally died at the age of 87 of a fractured hip.

In regard to the therapy, I am particularly interested in quinidine sulfate in large doses. In 1934, I had occasion to see some of the work of Wenckebach and Dressler, of Vienna, who were treating this condition with what I considered massive doses of quinidine sulfate. I was amazed to find they were giving 10 grains of quinidine sulfate by mouth every hour for twelve hours, if necessary.

I tried that when I came back to this country, with fear and trepidation, in certain selected cases. We cannot give it to any case where we have a weakened myocardium or a dilated heart, because there is always the danger of sweeping the mural thrombi out into the circulation and getting secondary emboli. But in certain selected cases, where the heart is not dilated, it is the ideal form of therapy.

We first test the patient out, as Dr. Schwab has told you, to see whether or not he is sensitive to the drug. We give him about three grains and then repeat it again in an hour. If there are no reactions or sensitization, and there is no evidence of cardiac dilatation, we give the large doses.

I have had eleven cases so far in which I have used 10 grains of quinidine sulfate every hour. I have never had to use it for the 12 doses Dr. Wenckebach suggested, because these cases, with one exception, responded by the end of the fourth hour, and that one responded at the end of seven hours.

Dr. Donovan C. Browne, New Orleans, La.—Will the essayist give the pharmacologic rationale for the use of quinidine in this type of tachycardia?

Dr. Schwab (closing).—The patient who has paroxysmal tachycardia of supraventricular origin will, in all probability, continue to have attacks from time to time. Some have them very frequently, while in others the seizures may be separated by long intervals.

Therefore it is of great importance that we educate these patients with reference to the physical measures which they, themselves, can use, and which will often successfully terminate a seizure. The reassurance the patient possesses by knowing he can do something himself which will terminate a seizure does a great deal toward making his life more comfortable. Accurate statistics are not available, but it is my belief that the majority of cases will respond to these physical measures and require no drug therapy.

I am sorry Dr. Luten did not amplify somewhat on the use of the cardiac glucosides in the management of intractable cases.

I believe the majority of failures which are encountered in the treatment of cases of paroxysmal tachycardia with quinidine are the result of the fact that dosage is inadequate. You see and encounter often the general feeling among the profession that quinidine is an extremely dangerous drug to use, and quite often in consultation when one advises giving it, even in the doses I recommended, we encounter considerable opposition. I have not employed it in the dosage which Dr. Hirsh mentioned but, in view of the successful results he has had, I think certainly it is worth trying.

As regards the rationale for the use of quinidine in patients with paroxysmal tachycardia of supraventricular origin, I am not certain I can answer that question. As yet we have inadequate information as to the nature of the biochemical disturbance in the heart muscle which causes a certain portion of it to become hyperirritable and initiate a paroxysm. There are some who feel that perhaps paroxysmal tachycardia is due to circus rhythm, such as we know to be the basis for auricular fibrillation and possibly ventricular fibrillation. If this is the case, the rationale for the use of quinidine is

obvious. We know also that quinidine influences the conduction time and refractory period of auricular muscle. Most certainly these factors are disturbed in instances of ectopic mechanism disturbances of this type.

THE TRANSMISSION OF *ENDAMEBA HISTOLYTICA* AND AMEBIC DISEASE*

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My reason for discriminating in this title between the transmission of a parasitic organism and the disease it causes is that the production of amebic disease is not necessarily the immediate and perhaps not the inevitable consequence of amebic colonization in the bowel. In his brilliant monograph entitled "Amebiasis and Amebic Dysentery," Col. C. F. Craig[1] says (p. 26):

"In most individuals harboring this parasite * * * the small lesions produced * * * heal quickly and no symptoms of the infection occur, or the symptoms that do occur are so slight as to be overlooked or regarded as due to other causes."

Thus, in an analysis of the transmission of *E. histolytica,* we are concerned only with those circumstances that make possible the transfer of infective organisms from one person to another, whereas, in a discussion of the epidemiology of amebic disease, we must inquire as well into those factors which cause the amebic invasion to become symptomatic.

It must be admitted at the outset that our knowledge of these matters is meager and unsatisfactory. Protozoologically, the organism is not an easy one with which to work. Accuracy in microscopic identification comes only with careful training and much practice. The organism was not cultivated *in vitro* until 1925[2] and has not as yet been continuously cultured free from other micro-organisms for periods exceeding 14 days.[3] Completely satisfactory experimental animals have not been readily available. Serologic tests have yet to acquire the precision and dependability that are characteristic of their application with many other host-parasite systems.[4 5] Each year sees improvements in diagnostic technic, but this is naturally at the expense of uniformity and comparability of the information acquired during the developmental period.

Epidemiologically, the classical difficulties and limitations of the science beset the student of amebiasis. Evidence is largely indirect and *ex post facto.* Observations cannot be controlled but must be equated against normal probability. Conclusions are correspondingly provisional and inferential.

Fortunately, these hindrances have not kept investigators from exploring the mechanics of *E. histolytica* transfer nor of the factors and circumstances which predispose toward clinical manifestations. The object of this paper is briefly to summarize and interpret some of their more recent and significant findings.

TRANSMISSION OF *ENDAMEBA HISTOLYTICA*

Since the turn of the century, the following protozoologic propositions concerning transmission have been generally accepted: (1) that the only natural way of acquiring amebic infection is by swallowing the encapsulated, resistant, spore-like cyst of *E. histolytica* and (2) that the ultimate source of such cysts is the feces of infected persons who are free from acute symptoms at least temporarily. It is, therefore, important to see how long these cysts are able to remain infective outside the human body under natural conditions, how numerous the cyst-passing individuals are in our native populations and their geographic and demographic distribution.

Effect of Environmental Influences on Cysts.—The environmental influences most unfavorable to these stages are drying and high temperatures. Amebic cysts are much more susceptible to desiccation than are bacterial spores. Several investigators have observed that they die within five to ten minutes when dried in the air.[6 7 8] Dust, therefore, can hardly be a medium of transmission and some have maintained that contaminated fingers are correspondingly unimportant.[9 21]

The best measurements of their thermal tolerance indicate that the cooler it is, the longer the cysts can live. The most precise information on this point is available from a study recently reported by Chang and Fair.[10] Exposing cysts in water at different temperatures until positive cultures could be recovered no longer, it was shown that the effect of temperature on survival time was in accord with the known viability-temperature relationships of primitive organisms. Cysts

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at freezing temperatures lived for nearly three months, their life-span being reduced by two-thirds for each successive ten-degree (Centigrade) rise in temperature. Thus at 10° C. (50° F.), they lived one month; at 20° C. (68° F.), 10 days; at 30° C. (86° F.), 3 days, and so on.

Yorke and Adams[11] have shown that these organisms can withstand a five-minute exposure to 45° C. (113° F.) but not to 50° C. (122° F.). They also demonstrated that cysts lived longer at freezing and laboratory temperatures in water than in undiluted feces. Thus we may conclude that foods and drinks heated to 122° F., or beyond, may be dismissed from consideration as possible vehicles of infection.

Incidence and Distribution of Cyst-passing Individuals.—In the last thirty years, and especially since the first World War, numerous surveys have been made in various parts of the United States to determine the incidence of intestinal parasites in general and of *E. histolytica* in particular. Due, however, to variations in the condition, type and number of stools per person examined, in the methods of examination employed and in the size and composition of the population samples investigated, the results of these surveys cannot be displayed side by side to give an accurate, nationwide picture of the distribution of amebic infection. Since 1926, Craig[12] has maintained that from 5 to 10 per cent of the general population harbor the parasite, an estimate that seems to be in accordance with the known facts and one which has been generally accepted. In 1940, the apparent incidence in 60,491 individuals examined in 26 surveys in various parts of the country was 9.8 per cent.[1 3]

It is evident from the following representative surveys that the incidence in the South is higher than in the North and that it is higher in residents of unsanitated rural areas than in urban populations. For example, in an isolated, farming community of 75 families in north-central Tennessee, Milam and Meleney[14] found 38 per cent of those examined positive for *E. histolytica* by a diagnostic method which, they felt, revealed 50 to 60 per cent of the actual positives. In contrast, a survey of 20,237 residents in the same state, similarly examined, revealed a carrier rate of 11.5 per cent.[15] In New Orleans, Faust and Headlee[16] reported the amebic incidence in 4,270 white out-patients to be 8.3 per cent which, corrected for age-distribution, was raised to 13.2 per cent. Suburban infection rates were higher than in the urban sections.

In comparison with the prevalence just noted for the South, Reed and Johnstone[17] found the amebic rate among 1,000 prisoners in California, most of whom were new admissions, representing all social and economic strata, to be 9.2 per cent.

In the East, Wenrich, Stabler and Arnett[18] examined students entering a professional school mainly from Philadelphia and New Jersey. An incidence of 4.1 per cent was found.

Based on these and other investigations too numerous to be catalogued here (see [1 13 19]), the author is of the opinion that the incidence of *E. histolytica* probably ranges from 5 to 10 per cent in the northeastern and central parts of this country, 10 to 15 per cent on the West Coast, and 15 to 25 per cent in the South.

As is well illustrated in the Tennessee[15] and Louisiana[16] investigations, the incidence of *E. histolytica* is very low in young children. By the time they have reached school age, however, their percentage of infections has increased to a level that is maintained more or less consistently thereafter throughout life.

Variations in the occurrence of cysts in the two sexes are not so marked in this country as in some others[20] where a differential exposure to infection accounts for the disparity. Faust and Headlee[16] report an exception to this general rule in the higher amebic rates observed in adult females than males in New Orleans.

The best evaluation of the comparative incidence of cysts in negroes and whites living under comparable conditions in this country is available from the Tennessee study.[15] No significant difference between the prevalence of cysts in the two races was found.

Endemic vs. Epidemic Amebic Infection.—Most amebic infection is endemic, that is to say, its incidence does not ordinarily vary from year to year outside of anticipated seasonal fluctuations. Epidemic occurrences are rare. Indeed, as pointed out in the official report[21] of the amebic epidemic in Chicago (p. 3), the prevailing opinion of authorities prior to the Chicago episode was that epidemic amebic infection did not exist at all. Since 1933, this view has been altered. Nevertheless, the conditions under which epidemics occur are so special that their transmission is discussed separately.

Modes of Transmission of Endemic Amebiasis.—Cool water is certainly an ideal medium for the

transmission of *E. histolytica* cysts. It provides dilution of the stool, moisture and temperatures compatible with survival for considerable periods of time. The presence of chlorine in amounts customarily employed for water treatment is not injurious[10 22] to the cysts. Water is probably an especially important vehicle of transmission in unsanitated areas. Certainly every opportunity is offered where shallow wells, improperly curbed, permit the entrance of polluted surface-water run-off. Cysts settle to the bottom, but the usual bucket-and-chain equipment probably stirs up the water sufficiently whenever it is lowered into the well to resuspend the organisms in it.

Other presumptive evidence of the role of water in the transmission of amebic cysts is supplied by Clark[23] who recalls that, prior to 1914-15, when the modern water system of the Panama Canal Zone was put into operation, an incidence of 4.25 per cent of amebic disease was noted in 4,000 autopsies; the incidence in the next 2,800 autopsies was 0.57 per cent. Hegner[24] has made a similar observation in Guatemala. While such demonstrations do not prove the transmission of amebic organisms by contaminated water, they do strongly suggest its probability.

Any moist food which is eaten cold, especially if it has not been cooked or pasteurized, offers potential facilities for the transfer of amebic cysts from the soiled fingers of one individual to the intestine of another. That possibility is enhanced in the case of leafy and rooty vegetables eaten raw, such as lettuce, celery, endives, radishes, and carrots, if they have been fertilized with human excrement or "freshened" for marketing purposes with contaminated water. It is probable that contact with such foods is the cause of the high rates of amebic infection reported from certain food-handler groups.[7 25 26]

This was well illustrated in a survey made by the author[26] in an unsanitated mining community in Mexico. Most of the vegetables consumed in the town were raised in a small, outlying truck-gardening section. This was operated by Chinese who irrigated the plots with the town sewage purchased because of its water content. The roots of the plants were, therefore, literally bathed in human excrement. The people living in that area, most of whom were concerned in the gardening enterprise, had the highest amebic-cyst incidence (44.1 per cent of 34 individuals) of any group examined. That the handling of these vegetables in the consumers' households led to infection of the domestics seemed strongly indicated by the fact that the group of 51 individuals so employed showed an infection rate of 35.1 per cent, whereas the incidence in the entire population examined (2,303) was 12.9 per cent.

The positive role of the food-handler in the dissemination of *E. histolytica* cysts, vigorously championed by Colonel Craig, is the subject of much controversy. The affirmative stand is based on the logical assumption that, unless infected food-handlers are scrupulously clean in servicing themselves at the toilet and immediately washing their hands thereafter, they may convey cysts from their soiled hands to cold, moist foods such as salads, uncooked seafoods, dairy products, sandwich spreads, and thence to the consumers of these foods. It has been shown[27] that *E. histolytica* may be retained under the nails of unwashed hands for somewhat longer periods than on the palmar surface of the fingers.[8] Spector and her associates[28] were successful in recovering cysts from the hands of 5 out of 74 carriers. In further support of this carrier contact thesis, numerous instances are cited[29 30 31] of the multiple occurrence of similar assortments of intestinal protozoa in families living under sanitary conditions where it is hard to see how transmission could have been effected except by this mechanism.

Prior to the Chicago epidemic, this argument was virtually unchallenged. The final demonstration that this outbreak was not due, as at first supposed, to the activities of food-handlers, plus Spector and Buky's verification[8] of the previous findings of others concerning susceptibility of cysts to drying, led some to believe that food-handlers were rarely, if ever, concerned in the spread of amebic infection.[21 32] This point of view has been strongly presented by Sapero and Johnson,[9] who have found that exposure of naval personnel to presumably infected food-handlers in Central America and to known infected food-handlers in naval messes did not result in amebic incidences higher than those of new recruits. They contend, therefore, that the failure of transmission under these favorable conditions indicates that such a method of dissemination is probably unimportant and that widespread reforms directed against food-handlers in the United States are not justified.

The carriage of *E. histolytica*-cysts by flies has been considered to be of importance ever since Thomson and Thomson[33] demonstrated in 1916 that the organisms could be found in the intestinal contents and dejecta of the insects. This observation has been confirmed in essence by numerous other investigators, including the Ten-

nessee investigators.[34] While the normal appearance of amebic and other protozoan cysts recovered from the fly gives every reason to believe that these organisms are viable, their actual survival in or on these insects has never been proved by critical tests and so their significance in transmission remains presumptive rather than demonstrated.

The possibility that lower animals act as reservoirs for human amebiasis has been well investigated. Organisms identical in appearance with *E. histolytica* have been found on rare occasions in dogs[35 36] and rats[37 38 39 40] and in their excrement. In a survey of more than 2,500 rats in Baltimore, Andrews[40] showed that the likelihood of human infection from this source is virtually negligible. Frye and Meleney investigated pigs, rats, mice, chickens and dogs in Tennessee, but obtained no evidence that they were contributing to human amebic infection.

Modes of Transmission of Epidemic Amebiasis.—As far as is now known, epidemic prevalences of amebic disease are due to the operation of one or more of the factors usually concerned with endemic transmission suddenly exaggerated in significance by some unusual circumstance. In two of the three epidemics described in this country, water has been involved; the third was believed to be due to transmission by flies.

The epidemic focus in the first Chicago experience[21] was localized in two hotels and the unusual circumstances which led to the infection of their guests were: (1) plumbing cross-connections between sewerage and water lines which allowed the circulation of sewage-contaminated water to hotel rooms; and (2) a rotting wooden plug in an overhead sewer which permitted leakage of raw sewage into an open cool-drinking-water tank below. As a result of these plumbing defects, the consumption of massive dosages of cysts by persons drinking water at these hotels was inevitable and a calamitous epidemic of amebic disease resulted.

The second epidemic reported from Chicago[41] occurred in 1934 at a stockyard fire and involved not only amebic but other acute enteric infections as well. Many of the firemen and spectators who drank water from pipes running to watering troughs experienced one or another of various intestinal upsets, including amebic diarrhea and dysentery. It was later shown that this water was from a private system owned by the stockyard concern. It ordinarily issued from a deep well but, as the pump had been removed for repairs, a supply from a nearby sewer, ordinarily chlorinated and filtered, was drawn on to assist in the fire-fighting. On the day of the fire, chlorination and filtration were not carried out and the enteric epidemic resulted accordingly. Firemen who drank only from municipal water supplies were not affected.

The third epidemic in this country, which antedates the other two, was described by Craig.[42] It occurred in 1916 in military troops quartered at El Paso. It did not involve civilian populations who used the same water supplies as the troops. The incidence of infection was correlated with the relative number of flies. The companies most seriously affected were mounted organizations which, by reason of their horse populations, had the greatest number of flies. The height of the epidemic occurred just after the maximum prevalence of flies. Sickness ceased when the flies disappeared in the fall.

FACTORS CONCERNED IN THE PRODUCTION OF AMEBIC DISEASE

Relative Incidence of Amebic Disease and Amebic Infection.—Little is known about the prevalence of amebic disease in relation to amebic infection. This is due more to inadequate morbidity reporting than to lack of protozoologic information. Clinical cases go undiagnosed, some are misdiagnosed and many of those diagnosed are not included in official vital statistics. The most that can be said is that the ratio of cases of frank amebic abscess, dysentery or diarrhea to the known prevalence of amebic parasitism is very small, probably not more than a few per cent.

To these relatively well-defined complaints attributable to amebae, must be added an unknown number of cyst-passing individuals who report vague abdominal distress, gastro-intestinal discomfort, irritability, loss of weight, low-grade secondary anemias, and so on, without a clear-cut picture of organic disease. As noted by Craig,[43] similar symptoms may occur in other disease conditions of the gastro-intestinal tract so that this complex should be viewed only as suggestive of amebic infection.

Some instances of combined parasitism and sickness occur, furthermore, in which it appears that the presence of amebae is only incidental and not at all concerned with the production of these symptoms. For example, it was shown in Baltimore that several gastro-intestinal patients with *E. histolytica* in their stools but without diarrhea, dysentery or demonstrable organic disease failed to improve as a result of amebicidal

treatment, but responded readily to antispasmodics, hypnotics and sedatives. When those pharmacotherapeutic measures were discontinued, the symptoms reappeared though the amebic infection had been eradicated.

Craig[43] has stated that there is no such thing as a "healthy" carrier of *Endameba histolytica*, a belief which apparently contradicts the experience and conclusions of many other protozoologists. I suspect that this is largely a question of definition. In elaborating his views, Craig goes on to say that his use of the term refers to individuals "in whom no lesions caused by the parasite are ever produced," and that "in every individual harboring this parasite, it is a potential source of injury to the tissues and health of the host." While internists encounter individuals passing large numbers of *E. histolytica* cysts but in whom intensive clinical study supported by all the diagnostic facilities of a large hospital fails to reveal any subjective or objective deviation from normal health standards,[44] it is improbable that any of them would be willing to assume that these persons would never thereafter experience difficulties due to *E. histolytica* or, on the basis of such an assumption, to leave the parasitism untreated. Why is it that a small minority of persons harboring *E. histolytica* experience severe symptoms and others only minimal or no discomfort? While[9] categorical answers cannot be given to this question, some information pertaining to the question is available and is briefly summarized herewith.

Climate.—Frank amebic disease occurs more frequently in the tropics and sub-tropics than in temperate zones; in temperate zones, it is more common in the warmer months of the year than in the cooler months.[45] It is well recognized by tropical clinicians that persons with stubborn, intractable amebic dysenteries, quickly benefit by a return to temperate climates or higher altitudes where the weather is cooler. Whether the higher incidence and intensity of symptoms correlated with high atmospheric temperatures and humidities are due more to enhanced exposure to infection or to reduced host-resistance is not known; probably both factors are concerned.

Virulent and Avirulent Strains of E. Histolytica.—Many observers feel that certain strains, some of which are distinguishable by size, of *E. histolytica* vary markedly in their pathogenicity. Several authors, including Brumpt,[46] have even suggested separate specific status for the so-called "small race" which are regarded by some as avirulent. While I have seen dozens of human infections with the small race of *E. histolytica*, I cannot recollect that I have ever seen it associated with dysentery. Spector[47] in reviewing her experience in Chicago with large and small amebic races, concluded that they were culturally, immunologically, clinically, and pathogenically distinct, both in lower animals and man. The small race produced only mild symptoms, no abscesses and no evidence of intestinal ulceration. Simic has worked in Jugoslavia with strains of amebae that resembled E. *histolytica*, but which were non-pathogenic to man or kittens. Whatever the virulence of the small race for man, it seems to be capable of producing dysentery in kittens.[49 50 51]

The most illuminating studies to date on the subject of differential virulence are those of Meleney and Frye in Tennessee.[50 52 53] In carefully controlled experiments with large numbers of kittens, they have shown that variations in virulence are intrinsic characteristics of *E. histolytica* and that, in general, these attributes are retained with relative consistency during years of artificial cultivation. Though the degrees of pathogenicity varied, every strain studied was capable of producing lesions in kittens.

Evidence of increase or decrease of virulence in experimental animals is conflicting, but the more recent observations agree that direct animal passage tends to intensify the pathogenicity of strains of *E. histolytica*.[53 54 55] Cleveland and Saunders[54] thought the enhanced virulence was due to corresponding changes in the accompanying bacterial flora, an opinion in which Spector[57] concurred. The Tennessee investigators,[56] however, incline to the belief that the augmented pathogenicity is due to the increased multiplicative, mechanical and histolytic activities of the parasites in a living host which offers more favorable surroundings than the culture tube. How dependably these conclusions, based on observations of *E. histolytica* in lower animals, reflect the behavior of this parasite in man remains to be seen.

Diet.—Some years ago Hegner[58 59] showed that it was possible to reduce the number of certain intestinal protozoa in several types of hosts by feeding a diet high in animal protein which resulted in shifting the predominance of the bacterial flora from sugar-fermenting to proteolytic types.[60] More recently, Faust and his associates[61-64] have demonstrated the favorable influence of liver products and the unfavorable

effects of "ventriculin" and canned salmon in modifying the course of experimental amebic infections in dogs. This presumably is due to the action of these substances in accelerating or depressing the resistance of the intestinal tissues against amebic attack. This suggests that the dietary habits of individuals or a population may have a profound influence in adjusting the balance between ameba and host.

Natural and Acquired Immunity.—In spite of equivalent exposures in family or community environments that offer every likelihood of infection, some individuals show no sign of harboring the parasite. Whether these persons are naturally immune or just the beneficiaries of chance, has never been determined.

Most observers agree that populations in which amebiasis is highly endemic tend toward the so-called "carrier" or chronic state, while newcomers of any age promptly exhibit acute symptoms of amebic disease. The resistance of the native in contrast to the susceptibility of the foreigner suggests that the local population may have acquired an immunity. Likewise, as Craig[65] points out, some individuals recover spontaneously from the infection. Whether these phenomena are evidences of an immunizing process stimulated by the absorption of amebic antigen or the protective effects of certain food substances has not been investigated to my knowledge. It is well-known that serologic antibodies can be identified in the blood of many infected persons.[66]

SUMMARY

In summarizing this discussion, we may say that the reservoirs of amebic infection are all about us. They probably constitute from 5 to 10 per cent of the population, being most numerous in the South, least so in the northeastern region of this country. As far as is known, transmission occurs most frequently by means of polluted water supplies, insanitary excreta disposal, personal contact with carriers, insect conveyance and contaminated foods. Epidemic transmission may be due to water or flies.

Factors that may be concerned in the production or suppression of symptoms appear to be temperature, strains of *E. histolytica* that are intrinsically more or less virulent than others, rapid passage from one host to another, dietary effects and immunity acquired by experience with the infection.

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Discussion follows paper of Drs. Schulze and Ruffin, page 705.

THE CLINICAL ASPECTS OF AMEBIC DYSENTERY AS SEEN IN NORTH CAROLINA*

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In an earlier publication[1] attention was called to the prevalence of amebic dysentery in North Carolina. The frequency with which this disease has been encountered since then; the ease with which the diagnosis can be made; and the gratifying response to proper treatment has led us to feel that a further report on the clinical aspects of this subject might be of interest.

Material.—This report is based upon a study of 95 cases of proven amebic infection, 54 of which have been reported previously.[1] The dis-

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tribution by age, race, and sex is shown in *Tables 1* and *2*. Of the entire group of 95 patients, 89 had an active diarrhea; the remaining had liver or lung abscesses, with no history of disturbance of bowel function that could be elicited. Of the 89 patients having an active diarrhea, 13 had complications or coexisting diseases, as shown in *Table 3*.

History.—The symptoms of the 76 patients without complications were studied in detail, and are shown in *Table 4*. The onset of the diarrhea was gradual in most cases (71 per cent), though 18 patients (23 per cent) reported a sudden onset. All of the 76 patients observed gross blood in the stool, and most of them (82 per cent) noted the presence of mucus. Pain in the abdomen and tenesmus were common though not severe. Weight loss, while not invariable, was marked in many cases, varying from 15 to 20 pounds. Fifteen patients continued their usual work, but 22 (29 per cent) were incapacitated because of their disease. Anorexia, nausea and vomiting were present in a small percentage of the cases. The number of stools varied from five to twenty-five per day. In only three patients could a history of a similar condition existing in the family be obtained. In 64 patients, nothing could be discovered from the histories which would suggest a predisposing factor. However, in nine patients, possible precipitating factions were pregnancy, influenza and operations.

The course of the disease was chronic. In many instances it had existed for several years before the patient was first examined in this clinic. However, several patients were seen after having had diarrhea for only a few days or weeks. In 21 of the 76 patients, a history of spontaneous remissions was reported. Ten had been treated previously with temporary subsidence of symptoms. All of the 76 patients had an active diarrhea when first examined.

Physical Examination.—Except for weight loss and occasional abdominal tenderness, the

AMEBIC DYSENTERY

DISTRIBUTION OF CASES BY AGE, RACE AND SEX

	0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79
Number	5	3	28	20	16	17	5	1
Percentage	5.2	3.2	29.5	21.1	16.8	17.9	5.2	1.1

Table 1

	Male	Female	White	Colored	Total
Number	79	16	85	10	95
Percentage	83	17	89	11	100

Table 2

AMEBIC DYSENTERY

1. Patients having active diarrhea		89
(a) Uncomplicated	76	
(b) With complications liver or lung involvement	6	
(c) With coexisting diseases	7	
(1) Idiopathic ulcerative colitis	5	
(2) Typhoid fever	1	
(3) Carcinoma of stomach	1	
2. Patients without history of diarrhea		6
(a) Liver, lung or brain abscess	5	
(b) Intestinal obstruction	1	
Total		95

Table 3

SYMPTOMS OF 76 PATIENTS WITH ACTIVE DIARRHEA AND NO COMPLICATIONS OR COEXISTING DISEASES

	Number	Percentage
Gross blood in stools	76	100
Mucus	62	82
Onset of diarrhea		
Gradual	54	71
Sudden	18	23
Abdominal pain		
Present	49	64
Absent	13	17
Weight loss		
Present	42	55
Absent	7	9
Disability		
Present	22	29
Absent	15	19
Anorexia		
Present	18	23
Absent	19	25
Nausea and vomiting		
Present	16	21
Absent	25	33

Table 4

routine physical examination was not significant. In the majority of the 76 patients, the temperature ranged from 37 to 37.5° C., with a normal pulse rate. In ten patients the temperature reached 38° C. or above, and in four of this group the temperature rose above 39° C., even though there was no clinical evidence of liver or lung involvement.

Proctoscopic Examination.—Proctoscopic examination was performed in 61 of the 76 uncomplicated cases. Ulcers were observed in 52 patients (85 per cent). These ulcers were usually shallow, multiple and discrete, with undermined edges, varying from 2 to 5 mm. in diameter, the intervening mucosa being slightly edematous, but otherwise normal. They were frequently covered by a yellowish gray exudate or mucus which almost invariably contained motile trophozoites. These were present in 82 per cent of the cases which were examined proctoscopically (*Table 5*). It should be emphasized that the proctoscopic picture in amebic dysentery as described above is entirely different from that usually seen in chronic idiopathic ulcerative colitis, in which the mucosa is diffusely inflamed and granular with pin point ulcers.

Laboratory Findings.—Anemia was uncommon, only eight patients having a hemoglobin of less than 70 per cent, and sixteen a red count of less than four million. A leukocytosis was present in over 60 per cent of the cases and there was also a definite increase in the polymorphonuclears in 60 per cent of the patients. It is interesting to note that an eosinophilia may occur (*Table 6*).

PROCTOSCOPIC EXAMINATION IN 61 PATIENTS HAVING ACTIVE DIARRHEA

	Typical Ulcers		Trophozoites	
	Present	Absent	Present	Absent
Number	52	9	50	11
Percentage	85	15	82	18

Table 5

BLOOD FINDINGS IN 76 PATIENTS WITH ACTIVE DIARRHEA AND NO COMPLICATIONS OR COEXISTING DISEASES

Hemoglobin

	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-109
Number	1	0	2	5	18	15	19	7
Percentage	2	0	4	7	27	22	28	10

Red Blood Count

	2-3 Million	3-4 Million	4-5 Million	5-6 Million
Number	3	13	28	11
Percentage	5	24	51	20

White Blood Count

	3,000-4,000	4-6	6-8	8-10	10-12	12-15	15-20	20-25
Number	2	4	8	7	17	11	7	2
Percentage	3	7	14	12	30	19	12	3

Polymorphonuclear Cells

	40-49	50-59	60-69	70-79	80-89	90-99
Number	1	2	18	19	11	1
Percentage	2	4	34	37	21	2

Eosinophils

	0-1	2-3	4-5	6-8	9-12
Number	26	9	9	6	1
Percentage	51	18	18	11	2

Table 6

INVOLVEMENT OF COLON AS SHOWN BY BARIUM ENEMA IN 31 PATIENTS HAVING ACTIVE DIARRHEA

	Normal	Entire Colon	Cecum Alone	Descending and Sigmoid	Cecum and Transverse	Cecum and Descending	Transverse Alone	Rectum Alone
Number	7	8	7	4	2	1	1	1
Percentage	22	26	22	13	7	3	3	3

Table 7

Barium enema examination was performed in 31 patients and recorded as being normal in seven, or 22 per cent. The involvement of different portions of the colon is shown in *Table 7*.

Diagnosis.—The diagnosis in those 89 patients having an active diarrhea was based upon the demonstration of motile amebae containing red cells in all but six cases. In two of these, a rectal mass was observed, biopsy of which showed amebae in the microscopic section. The remaining four cases had typical ulcers which healed promptly after specific therapy. In four patients the proctoscopic findings were negative, but trophozoites were demonstrated in the mucus obtained by proctoscopy. In the six patients without a history of diarrhea (*Table 3*), the diagnosis was established at autopsy. Patients having cysts only in their stools without the characteristic ulcers or trophozoites were purposely excluded from this series.

Treatment.—Of the 76 patients having an active diarrhea without complications or coexisting diseases, 67 were treated with different drugs or combination of drugs, the results of which are shown in *Table 8*. The dosage usually employed was: carbarsone 0.25 gram twice daily by mouth, chinofon 5 grams by retention enema, emetine hydrochloride 64 mg. subcutaneously, and "diodoquin" 2 tablets three times a day orally. The treatment was continued for ten days. The result of this treatment was usually dramatic, regardless of the method used, though it was our impression that the best results were obtained when the combination of carbarsone by mouth and chiniofon by retention enema was given. In general, there was a marked change in the patient's sense of well being after 24 hours, and of the 67 patients who were treated, the diarrhea subsided after two days of treatment in 39, or 60 per cent of the cases. On proctoscopy the ulcers showed evidence of healing within a few days after therapy was instituted and were almost completely healed after ten to fourteen days. The mucous membrane of the rectum and sigmoid in these patients was entirely normal so far as could be determined by proctoscopic examination. The trophozoites disappeared from the stools following the usual therapy in the seven patients having coexisting diseases (*Table 3*). The average hospital stay of the 76 uncomplicated cases was fourteen days.

Liver and Lung Involvement.—Eleven patients had liver or lung involvement. In four of these, a diagnosis of hepatitis was made, based upon the presence of motile amebae in the stool, a large tender liver, and a clinical picture of sepsis. All these responded promptly to the administration of emetine. Two patients had liver abscesses which were treated with emetine, but surgical drainage was also required. Both recovered completely. Two patients had lung abscesses with trophozoites in the sputum and recovered after emetine therapy. In three patients with liver and lung abscesses, the diagnosis was made at autopsy.

TREATMENT OF PATIENTS HAVING ACTIVE DIARRHEA WITHOUT COMPLICATIONS OR COEXISTING DISEASES

Medication	Recovered	Improved	Unimproved	Died	Total
Chiniofon	6			1	7
Carbarsone	12	1	1		14
Carbarsone and chiniofon	33	1			34
Emetin and chiniofon	3		1		4
Carbarsone, emetin and chiniofon	2		1		3
Carbarsone and emetin	4				4
"Diodoquin"			1		1
Total					67

Table 8

Follow-Up. — Information concerning their course after discharge from the hospital was obtained in 44 patients of the group of 76 uncomplicated cases. The results of this follow-up study are shown in *Table 9*. As can be readily seen, there was no relationship between relapses and the drug used. It is significant to note that 36 per cent of those patients who were followed had one or more relapses. One of these patients returned with a lung abscess, even though he had received adequate treatment and his diarrhea had subsided.

The following case reports illustrate the clinical course of a patient who responded promptly to therapy and remained well, and another who had frequent relapses in spite of treatment with many different drugs:

Case 1.—J. F. H., a 63-year old manufacturer, was first seen at Duke Hospital April 4, 1934. A diagnosis of benign prostatic hypertrophy was made, and on April 14 a transurethral resection was done. Recovery was uneventful. On September 27, 1934, he returned to the hospital with the complaint of bloody diarrhea. One month after operation he suddenly developed bloody mucoid diarrhea with much tenesmus and colicky abdominal pain. He had anorexia and lost ten pounds in weight. He averaged a stool every two or three hours. His diarrhea persisted until the time of his admission to the hospital in spite of various palliative measures.

Physical examination showed nothing of interest except slight generalized abdominal tenderness.

Hemoglobin was 13.5 grams (87 per cent); red blood cells were 4,720,000; white cells, 6,600; differential, polymorphonuclears, 51 per cent; eosinophils, 4 per cent; mononuclears, 8 per cent; large lymphocytes, 12 per cent; small lymphocytes, 25 per cent. Barium enema showed spasm of the cecum, but was otherwise essentially normal. Proctoscopic examination revealed many punched-out ulcers one to three mm. in diameter with normal intervening mucosa. The appearance was typical of that seen in amebic dysentery. Many motile trophozoites containing red blood cells were seen in a fresh preparation of mucus obtained directly from the ulcers.

He was given chiniofon 1 gram three times a day by mouth and retention enemas, 5 grams of chiniofon in 300 c. c. of water daily for ten days. His stools decreased from ten to three a day within the first forty-eight hours. Proctoscopic examination was repeated four days later and the ulcers were healing. No amebae were demonstrated in the smear made at this time. The patient was discharged after ten days of hospitalization.

On September 27, 1937, he returned to the hospital for general examination. There had been no recurrence of his diarrhea at any time and he had no other gastrointestinal symptoms.

He returned to the hospital in August, 1940, six years after the onset of his illness, for a follow-up study, reporting that he had had no diarrhea, nausea or vomiting at any time and no blood in the stools. Proctoscopic examination was entirely normal and there were no trophozoites or cysts in the stool.

Case 2.—R. T. B., a 30-year-old farmer, was first seen in Duke Hospital September 22, 1939. His present illness started August 2, 1939, at which time he had a sudden onset of bloody mucoid diarrhea of twelve to fifteen bowel movements per day. His diarrhea had continued until the time of admission to the hospital in spite of palliative treatment. He complained of anorexia, tenesmus and abdominal pain. There was no nausea or vomiting. He had lost weight, but the amount was unknown.

Physical examination was essentially normal except for moderate weight loss and slight abdominal tenderness.

Hemoglobin was 95 per cent; red cells were 4,410,000; white cells 11,000 with 74 per cent polynuclears, 4 per cent eosinophils, 9 per cent large lymphocytes, 12 per cent small lymphocytes and 1 per cent monocytes. Barium enema was essentially normal. Proctoscopic examination at the time of admission showed numerous punched-out ulcers with normal intervening mucosa. Innumerable motile trophozoites containing red blood cells were demonstrated from the proctoscopic smear. The patient was given 0.25 grams of carbarsone three times a day by mouth and retention enemas of 5 grams of chiniofon with 300 c. c. of water daily for ten days. In addition he was given thiamin chloride 5 mg. twice a day and nicotinic acid 25 mg. three times a day. Stools decreased from eight to one a day within forty-eight hours. He left the hospital after ten days of therapy with instructions to take another ten-day course of carbarsone two weeks after discharge. On October 30, 1939, he had recovered completely. Proctoscopic and stool examinations were negative.

On December 20, 1939, he returned, reporting that he had taken treatment for hookworm ten days before and following this had developed a diarrhea of eight te ten bloody mucoid stools per day. He was again found to have typical ulcers and motile amebae and was treated in the outpatient clinic, being given 0.25 grams of carbarsone three times a day for ten days.

He was next seen on January 10, 1940, having observed no improvement whatever after treatment with carbarsone. He was hospitalized January 22, 1940. Physical and proctoscopic examinations and stool findings were the same as on his first admission. Hemoglobin was 15.5 grams (100 per cent), red blood cells were 5,300,000, white cells 12,000 with 79 per cent polynuclears, 6 per cent large lymphocytes, 11 per cent small lymphocytes, 1 per cent eosinophils, and 3 per cent mononuclears.

FOLLOW-UP STUDY OF FORTY-FOUR PATIENTS HAVING ACTIVE DIARRHEA AND NO COMPLICATIONS OR COEXISTING DISEASES

Medication	Recovered	Relapsed
Chiniofon	2	1
Carbarsone	8	1
Carbarsone and chiniofon	15	10
Emetin and chiniofon		2
Carbarsone, emetin and chiniofon	2	
Carbarsone and emetin	1	2

Table 9

He was again given a course of carbarsone by mouth and chiniofon by retention enema for ten days with good results. He had a relapse in April, 1940, which was treated at home with a course of emetine and chiniofon with subsidence of symptoms. In June, 1940, he had another relapse which was treated again with carbarsone by mouth and chiniofon by retention enema with good results.

On August 5, 1940, he returned to the hospital for a follow-up study. Proctoscopic examination at this time was entirely normal. Smears were negative. On August 12, 1940, a barium enema was done which showed the cecum to be noticeably hyper-irritable and tender, the descending colon showing some spasticity.

One week later he had another relapse which was ineffectually treated with "diodoquin" (two tablets three times a day) until he was given nicotinic acid 100 mg. three times a day, following which he improved considerably. After ten days' treatment with carbarsone and nicotinic acid, he recovered completely.

Mortality.—Seven patients (7.4 per cent) in the whole series of 95 cases died (*Table 10*). Three patients without complications died. Two of these were diagnosed at autopsy and the third was moribund on admission. Three deaths were due to liver or lung abscesses, the diagnosis being made only at autopsy, and the other death was due to an intestinal obstruction. The last 40 patients seen here in the past three years have all recovered. Four of these had liver or lung involvement.

DISCUSSION

The purpose of this communication is to call attention to the prevalence of amebic dysentery in North Carolina; to the ease with which the diagnosis can be made in the majority of the cases, provided proctoscopic examination is employed; and to the prompt and dramatic recovery usually observed after proper treatment has been instituted.

The disease may occur at any age and is much more prevalent in the male. When there is active diarrhea, gross blood is almost invariably present (100 per cent in this series). Symptoms accompanying the diarrhea are apt to be mild, but occasionally these may be severe. The course of the disease is chronic and relapses are frequent. The physical examination usually is of little value in establishing the diagnosis. It should be emphasized that the two all-important procedures are proctoscopy and stool examination.

MORTALITY

	Number	Percentage
Whole series of 95 patients	7	7.4
76 uncomplicated cases	3	4
19 cases with complications	4	21
Last 40 patients (4 having complications)	0	0

Table 10

The ulcers alone are so characteristic as to justify diagnosis in most cases, and almost always mucus taken from the ulcer and examined immediately will reveal the presence of trophozoites. A warm stage is totally unnecessary when the stool is examined in this way. It should be remembered that trophozoites are rarely found in the solid stool and in stools containing large amounts of pus.

Other laboratory studies are of little value in establishing the diagnosis. The blood picture is not characteristic. Barium studies of the colon are sometimes suggestive but rarely, if ever, justify diagnosis.

The diseases which are most apt to be confused with amebic dysentery are ulcerative colitis, bacillary dysentery, polyposis, carcinoma, and tuberculosis of the colon. It should be realized, of course, that these diseases may actually coexist in any patient. In general, accurate diagnoses can be made, but occasionally one has to rely upon the response to specific therapy.

Complications were frequent and usually serious, 12 patients (13 per cent) having liver or lung abscesses. It should be pointed out that these abscesses may develop in the absence of diarrhea or history of diarrhea. One patient, successfully treated for his diarrhea, subsequently developed a liver abscess.

Adequate treatment usually results in dramatic recovery. In our experience the administration of carbarsone by mouth and chiniofon by retention enema has proven most effective. However, in this series, relapses were common (36 per cent) regardless of the type of therapy used. The mortality of the whole series was only 7 per cent and there have been no deaths in the last forty patients treated.

It should be emphasized that this series of cases includes only those patients having active amebic colitis or liver and lung involvement. It is reasonable to assume that for every patient having an active amebic infection there are many who are cyst carriers and are asymptomatic. The prevalence of cyst carriers has been emphasized recently by Faust,[2] but the significance of these cysts is still debatable. Certainly their presence means tissue invasion somewhere in the course of the intestine, but this in itself appar-

ently does not constitute disease, or at least many such persons are symptom-free. The factor or factors which prevent the cyst carrier from developing the active disease are still unknown. In this series of 95 patients, only two could be found who had been subsisting on a well balanced diet. One of these developed her infection in the Chicago epidemic and another following an operation. The remainder were of the tenant farmer class and were known to have consumed a deficient diet. It is possible or even probable that a dietary deficiency is an important factor in the development of the active disease, and that it tends to result in relapses after recovery following proper treatment. It is significant to note that in this series, relapses occurred in 36 per cent of those patients who were followed, whereas Mateer[3] reports relapses in only 3 per cent, although the method of treatment was essentially the same in the two series. While this discrepancy may be due to difference in the virulence of the strain of the organism, still it is well known that there is a marked difference between the diet of the tenant farmer in the South and that of the laborer in the North.

Since the discovery of the *Endamoeba histolytica* by Lösch in 1875, much study has been devoted to its incidence, cultural characteristics, differentiation from other endamebae, method of invading the host and types of specific therapy. Relatively little is known concerning the individual factors which predispose its host to active disease. Equally little is known concerning the factors which cause relapses in some patients who have had repeated courses of antiamebic therapy. A thorough clinical and experimental study of this group of patients should be of material value in solving the problem of amebic infections.

CONCLUSIONS

(1) Amebic infections are common in North Carolina.

(2) Diagnosis of amebic dysentery by proctoscopy is simple.

(3) Amebic liver abscesses occur without previous diarrhea.

(4) Specific therapy is highly efficacious.

(5) Relapses (? reinfections) are common.

(6) A dietary deficiency may be an important factor in causing infection and relapses.

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DISCUSSION (*Abstract*)

Papers of Dr. Justin Andrews and Drs. William Schulze and Julian M. Ruffin

Dr. Henry E. Meleney, New York, N. Y. — Dr. Andrews has emphasized particularly the importance of water as a vehicle of infection and I believe that it is, under many circumstances, the most important vehicle of infection. In rural areas, where water purification is not practiced, it is probably more important than we can demonstrate and than one would think at first. I think also it is more important in certain cities than we have known. The example of Chicago has indicated the possibility of infections in hotels, and from the hotels that I have seen in some parts of the country, I believe that, hidden away, there are opportunities for infection. The difficulty is that people may not realize that they have become infected until they have passed on to other places and would naturally refer their infection to their present place of residence.

Flies also must be considered important. Our demonstration of *E. histolytica* in flies in Tennessee was not based on culture experiments and I do not think this has been done, but we worked with cockroaches also and demonstrated by culture that viable cysts could be recovered from the intestines and droppings, at least forty-eight hours after they had been fed. If this can occur in cockroaches, I believe it is true with flies also.

I believe that direct transfer of *E. histolytica* from one person to another is important under certain circumstances, particularly in families in rural areas and among people who have no sanitary sense. Mothers tending children and children playing with each other certainly offer opportunities for transmission, particularly where fecal disposal is bad. There is an illustration which Dr. Andrews did not mention, which indicates that this is possible. I refer to surveys which have been made in institutions for the insane. In Sweden and other countries it has been shown that the longer the patients stay in such institutions, the higher is the incidence of *E. histolytica*. This is undoubtedly due to bad personal hygiene and not to water supplies. It is less likely to be due to a carrier among the food handlers in the institution.

I wish to make a comment upon the pathogenicity of *E. histolytica* and the problem of whether infection of the intestinal lumen always means infection of the intestinal wall. From our studies in Tennessee we felt that there is a good probability that the ameba may sometimes live in the lumen without invading the tissues. If other intestinal protozoa can do this and if *E. histolytica* can live in a culture tube outside the body, some asymptomatic cases may be examples of simple lumen infection. This does not lessen the importance of treating every individual who has the infection, both to protect himself from invasion and to protect others from infection.

Dr. E. C. Faust, New Orleans, La.—In one sense Dr. Andrews' clear distinction between an infection with *Endamoeba histolytica* and amebic disease is as apt as

the distinction between hookworm infection and hookworm disease. The infection may be accompanied by no detectable symptoms whereas disease is a clinical entity. In another sense, however, the comparison is not applicable; amebae multiply within the bowel and thus may change a state of host-parasite stability of no clinical moment into an acute or chronic amebiasis of fundamental importance to the health or even the life of the patient. This not infrequently occurs when the patient's threshold of resistance is lowered by dietary deficiencies or indiscretions, worry, intercurrent infections and treatment, and probably several other causes.

The student of clinical amebiasis is interested not only in the more dramatic expressions of clinical amebiasis, as frank amebic dysentery, but also in the milder, more chronic types. He must also be aware of the grave dangers of asymptomatic amebic infection as the potential seed bed of the more acute expressions of the invasion of the bowel wall, of appendical involvement or amebic liver abscess. Thus, all patients harboring *Endamoeba histolytica* must be regarded as sufferers or potential sufferers from the tissue destruction produced by the parasite.

Dr. Andrews has clearly outlined the possible sources responsible for transmitting *E. histolytica* to man. Water probably plays the major role in major epidemics of amebic dysentery, but the common source of endemic amebiasis in communities has never been clearly demonstrated. There are probably several contributing causes, including grossly contaminated food and water, unclean food handlers, and filth flies, particularly in rural areas. One of my graduate students is just completing an investigation on the potential role of the fly in transmitting *E. histolytica* cysts. He has found that all species of filth flies tested ingest amebic cysts in human feces; that these cysts never hatch in the fly's gut, but are usually discharged in vomit drops or fecal deposits of the fly over a period of minutes to hours and remain viable, as demonstrated by their ability to excyst and grow in culture. Thus, the filth fly undoubtedly has the capacity to carry amebiasis from the human feces to food or water and should be regarded as a major danger.

Next time you are in a rural community and are sitting down to a bounteous repast where there are a lot of flies all around, just think of the possibility of those vomit drops and little fecal deposits.

I firmly believe that amebic infection is more common in the population than the 5-10 per cent incidence estimate of Colonel C. F. Craig (1934). My belief is based upon the greatly increased number of positives provided by the more recent enrichment technics applied to repeated stool examinations of the same patient, frequently doubling and at times even quintupling the incidence in a particular group, as compared with single stool examinations made by routine fecal film technics.

Amebiasis is a common infection in the United States and is responsible for considerably more undermining of health than is generally appreciated. The paper presented by Drs. Schulze and Ruffin provides evidence of endemic clinical amebiasis in a really substantial series of cases for the temperate zone, reminiscent of amebiasis in Tropical America. It is significant that all of these patients provided evidence of chronic amebiasis, apparently of considerable duration; that the great majority (89 of 95) had one or more dysenteric exacerbations; that eleven patients in the series had liver or lung involvement, and that of 31 patients to whom barium enemas were administered, 58 per cent had involvement of the cecum and 26 per cent had the entire colon affected. All of these data point to chronic amebiasis, with the cecum as the primary focus for most of the lesions. Furthermore, the neutrophilic polymorphonuclear leukocytosis in 60 per cent of the cases suggests secondary bacterial invasion of the intestinal lesions.

The care with which diagnosis was carried out in these patients is demonstrated by the combination of stool and proctoscopic examinations supplemented by barium enemas in 31 patients. It is of interest to inquire if stools were also cultured for *Shigella* infection, and whether x-ray plates suggested hepatic or pulmonary amebiasis.

The treatment carried out on the patients allows no room for criticism: emetin hydrochloride for the acute colitis and for the liver and lung complications; the more specific carbarsone and chiniofon (yatren) for prosecuting the eradication of the amebae from the bowel. The reinforcement of oral therapy with retention enemas of yatren was probably justified in these chronic cases, which are more resistant than early acute infections, in which the relapse rate would undoubtedly have been lower even without yatren by enema.

Dr. Joseph E. Knighton, Shreveport, La.—It was noted in one of the papers that we may see liver abscesses without diarrhea. I recall a patient I saw two or three years ago whose complaint was that of continuous low grade elevation of temperature with corresponding weakness. He had suffered with this condition over a period of several months.

When I saw him, of course, I made the usual general physical examinations and usual laboratory examinations, without finding anything to justify the presence of this continuous temperature, except the fact that I did find on physical examination a definite, well-marked, enlargement of the liver, which was quite tender. There was no history of diarrhea. He had never had diarrhea, but he had lived in the tropics, in the employ of an oil company over a period of a number of years.

In the absence of any other positive findings, I began vigorous treatment for amebic infection which, as suggested by these papers and discussions, consisted of emetin hydrochloride to begin with. The man's temperature promptly subsided and I had occasion to keep up with him over a period of several years. The hepatic enlargement disappeared promptly, all tenderness disappeared, and he had no further trouble. Of course, the treatment was followed up.

There is another case that comes to my mind that seems of special interest, which illustrates the power of these organisms to invade every tissue of the body. I recall a patient who had definite amebic colitis, had rather indifferent treatment, later had an amebic abscess of the liver, which ultimately was drained by a surgeon, went along, had treatment, and later came down with periostitis of the inferior maxillary. This persisted to the point apparently of the necessity of opening up and draining, and the pus from this showed very active, numerous amebic organisms.

That, I think, is just an illustration of how widely disseminated this organism may be.

Dr. John Tilden Howard, Baltimore, Md.—I should like to ask Dr. Andrews whether or not filtration of

city water supplies removes amebic cysts, that is, the ordinary filtration of city water through sand. I understand from him that no palatable water containing chlorine would kill the cysts.

I have a second question which I would like to ask Dr. Schulze, and that is about the doses of carbarsone which he gave to his carbarsone-treated patients. We have given after the manner of Hakansson rather larger doses than are usually recommended. I presume that the recommended dose is 0.25 gram twice or three times a day for ten days. We have given daily to 150 pound individuals seven or eight tablets or capsules, each 0.25 gram, and I have wondered about his dosage.

Dr. Andrews (*closing*).—I want to emphasize an opinion expressed in my paper to the effect that while there appears to be good evidence that *E. histolytica* can live in the human bowel for indefinite periods without clinical manifestations of disease, this is no reason for withholding anti-amebic treatment of individuals known to harbor the organism. All instances of parasitism should be regarded as potential cases and sources of infection for others. They should be properly and adequately treated.

It is a special satisfaction to me to note from Dr. Faust's discussion that the role of the fly in the transmission of *E. histolytica* has at last been definitely demonstrated. Experimental evidence of the viability of this organism in flies and cockroaches permits us to assign to these insects proper epidemiologic and control significance in the transmission of amebiasis.

Lastly, in response to Dr. Howard's question concerning filtration, it was shown originally by Mrs. Spector and her associates in the U. S. Public Health Service, that sand filtration of water supplies would completely remove amebic cysts and thus obviate the danger of their transmission in filtered water supplies.

Another method of treating water to render it potable in this regard, as shown by Chiang and Fair, is to hyperchlorinate it until amebic cysts are killed and then reduce the chlorine to the usual residual quantum.

Dr. Schulze (*closing*).—Dr. Faust wished to know whether x-rays of the chest were taken. Routinely x-rays of the chest were not taken; however, in all cases in which abscesses of the liver or lungs were suspected, flat plates of the abdomen and chest were taken and the liver was visualized with "thorotrast." Surgical drainage of liver abscesses was necessary in two patients but other patients with liver and lung abscesses responded to emetine alone.

Stool cultures for the dysentery group of organisms were not done in all patients.

In response to the query concerning the dosage of carbarsone: routinely we give 250 milligrams three times a day for ten days, and this course is repeated after a rest period of two weeks. Carbarsone is a drug that is not entirely innocuous and in the presence of liver damage, it is known to be quite dangerous; consequently, we never give it in cases in which hepatitis or liver abscesses are suspected.

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PHYSICIANS AND THE WAR

In the first World War, which the United States entered in 1917, the requirement for medical men was very much less than in the present emergency. What was considered the colossal scale of that conflict fades into something like camouflage in comparison with the program of the present. One may contrast the seven billion dollars appropriated in 1917 for conduct of World War I with recent billions under discussion in Congress for World War II and contrast the relative size of the armies and armaments and fields of battle. To name the engagement of twenty-five years ago a world war seems now merely a prophecy of things to come.

Medical needs of the present war will, of course, be expanded in proportion to all other equipment of material and men, but the number of physicians has fixed limits. They cannot be turned out on a conveyor belt by a mechanical genius, like bombing planes. The number of wounded in this war may be proportionately greater than in the first, because the weapons of offense are more devastating. The problem of supplies is already greater, increasing the possibility of food deficiency diseases in distant lands; and much of the battle ground is in tropical and subtropical countries, where infectious and parasitic diseases develop with greatest luxuriance.

One may add to this the fact that science itself has made tremendous strides in the past quarter of a century, and conditions may now be successfully treated which formerly had to be left to nature's hand. Medicine will have more soldiers to care for, and more work to do per soldier.

At the Atlantic City session of the American Medical Association in June, Paul V. McNutt,[1] Federal Security Administrator, addressed members of the Association upon the current requirement of the Government for physicians. His figures are of much interest. Up to now, nothing like enough men have been obtained by voluntary enlistment, which is far behind the figures for a comparable period in 1917. In the first six months of World War I, 12,000 physicians not obligated by reserve commissions volunteered. In the first six months of the present war, only 3,000 volunteers have been obtained, or one-fourth the number which volunteered in the same time twenty-five years ago. An army of nine million men is contemplated, with more than fifty thousand physicians. The armed forces will require 12 per cent of the male population; and 33 per cent of all living physicians, including retired men, or two-thirds of the men under the age of 45. Young men are needed, but older men are being accepted.

The Federal Security Administrator emphasized the need for physicians in industry as well as on battle fronts, and in defense areas, those communities which have mushroomed up from a village of a few hundred persons to a town of thirty to forty thousand; and of physicians for rehabilitation of the disabled.

World War I was said to be the first great war in which disease did not take a greater toll of soldiers than the enemy. The American medical corps of that war was the most efficient in history and the present generation will live up to that record and tradition. About twenty-two thousand physicians have been taken into service in the past eighteen months. Twenty thousand more will be required this year, and another ten thousand in 1943.

A well staffed medical corps is an essential cog in the war machine. The same rule which makes drafting the most equitable means of procurement of soldiers for combat must apply to

1. McNutt, Paul V.: The Urgent Need for Doctors. J.A.M.A. **119**:605 (June 20) 1942.

the personnel of the medical corps. They should be called by a special draft act. Otherwise the ultra patriotic of super age groups, or key men in industrial and teaching ranks, will be found volunteering and filling posts which would be better and more appropriately held by younger men.

CORPUS LUTEUM AND ABORTION

It is said that inevitably one-fourth to one-fifth of all human impregnations result in miscarriage. The Puritans believed that such mischances were a righteous punishment for conception on Sunday. This large abortion rate is generally considered to be nature's means of ridding the womb of defective fetuses. In a series recently studied pathologically, half the abortions were of abnormal fetuses. Only a third were due to maternal fault or disease.

If this is true, says Rutherford,[1] of Harvard Medical School, what is the use of attempting to treat threatened abortion? In many cases, treatment merely delays the casting off of a dead embryo. The average time of abortion is in the tenth week of pregnancy, while the average time of fetal death is in the sixth week. There is thus a period of a month or more in which the fetus has ceased to develop but function of the corpus luteum has not disappeared and the dead and degenerating products are retained. During this time, symptoms of threatened abortion may continue, and the patient may be treated over a considerable period and the normal emptying of the uterus delayed uselessly. Among these dead retained products of conception there is a tendency to hydatid degeneration, and a fair percentage of hydatid cases become malignant. Hormone and other treatment of a patient with a dead fetus is definitely contra-indicated. However, there is no clinical method for detecting the death of the ovum. One merely knows that it usually occurs some weeks before it is aborted.

Endometrial biopsies, says Rutherford, can throw some light upon the subject. They have been done accidentally during pregnancy without its interruption. He reports a considerable series done intentionally, with the idea of studying the decidual changes microscopically. He attempts to prophesy from these whether a threatened abortion is inevitable, the decidual picture which may improve under progesterone therapy, and the picture in which this has no place. He believes that he has obtained useful information by this means and that he has caused no abortions by his technic.

In the first trimester of pregnancy, in which most abortions due to defects of the ovum occur, the decidual microscopic picture in cases in which the fetus is not viable he considers rather clear cut, and a degenerating deciduum of this type he says is not favorably modified by progesterone therapy. The third month of pregnancy he calls the critical period, when the placental hormones are replacing the senile corpus luteum. At this time particularly certain cases of threatened abortion might be successfully treated with progesterone, which has a quieting effect upon uterine musculature. In cramping it may be useful. In cases of bleeding without cramps it may be harmful. The microscopic picture of the uterine lining in pregnancy he considers of definite prognostic value, and useful in determining the possible indications for progesterone.

Rutherford's investigations of the clinical effects of this hormone in abnormal pregnancy are most interesting. It is hardly desirable in the present state of knowledge that general use of uterine biopsy in threatened abortion be employed. However, physicians should recall that not all cases of threatened abortion or bleeding in pregnancy are benefited by corpus luteum, and that expulsion of a dead fetus should not be delayed by therapy.

1. Rutherford, Robert N.: The Significance of Bleeding in Early Pregnancy as Evidenced by Decidual Biopsy. Surg., Gyn. Obst., **74**:1139 (June) 1942.

GROWTH OF NERVE TISSUE

Pathogenic bacteria such as the gonococcus, Spirocheta pallida, filterable viruses and hemolytic streptococci, are inclined to require complex culture media for their growth. Blood is one of the materials frequently added to simple bacteriologic bouillons. Some bacteria require such vitamins as nicotinic acid. Higher single celled organisms like yeast utilize several growth factors, some of which are identical with the vitamins of human and animal nutrition. One of the growth-promoting factors of yeast is biotin, a sulfur containing product related to the group of B vitamins.

Hamilton and Plotz,[1] of the Army Medical School in Washington, have studied the effect of biotin upon cultures of nerve tissue. Their investigation was undertaken in the effort to im-

1. Hamilton, H. L.; and Plotz, H.: Use of Biotin for Stimulating Growth of Nerve Tissue and Other Cells *in vitro*. Proc. Soc. Exper. Biol. and Med., **50**:133 (May) 1942.

prove methods of cultivation of the viruses and rickettsiae *in vitro*. They note that very small amounts of biotin may have pronounced biologic effects. Under certain circumstances, biotin can increase the number of experimental tumors in rats fed a carcinogenic compound, butter yellow. They note that it stimulates the growth of nerve tissue in culture media. They hope to use the biotin nerve tissue cultures to produce a higher titer of virus and rickettsia in cultures for immunologic study.

Since all animals and vegetables have certain complex vitamin requirements, and bacteria similarly will rarely grow on perfectly simple media, it is to be expected that different tissues cultivated outside their normal habitat will require vitamin-like materials for growth. Chemists, physicists and bacteriologists are constantly breaking these requirements down to simpler elements. It is rather to be expected that different types of animal tissues outside the living body may require different growth factors, or at least different quantities of the accepted vitamins.

TWENTY-FIVE YEARS AGO

From Journals of 1917

World War I.[1]—American ships, flying American flags, have been sunk by German submarines. American citizens have been killed in the ruthless and inexcusable warfare on American commerce by a nation that disregards treaties and defies international law. There is no escaping the conclusion that Germany is waging war against the United States; and it only remains for President Wilson and Congress to declare that we are in a state of war with the Prussian monarchy.

Physicians in Service.[2]—The numerical strength of the Medical Department of our present standing Army, even before the declaration of war, was deficient; now that war is upon us and with the determination by our Government of equipping and training an army commensurate with the dignity and rank of this Nation, there will be needed from 20,000 to 30,000 medical reserve officers.

Food for War.[3]—Perhaps the most serious danger lies in the scarcity of meats and other proteins. * * * It is said that since meats and fats have almost disappeared from the German dietaries, tuberculosis and other diseases have increased enormously.

The housewife in cutting down her household expenses, particularly her grocery bills, and in raising vegetables in her backyard, may serve her country as well as her husband or son who is called to the colors * * *

Buy Liberty Bonds.[4]—The appropriation of $7,000,000,000.00 for conducting the war is the largest amount ever provided for any purpose in the history of the world. * * * The younger men, except for unusual reasons, should enlist, but many of the older men cannot. The older men * * * are better able to buy bonds than their younger confreres. * * * Doctor, buy Liberty Bonds today.

Medical Schools.[5]—France and England made the mistake of closing some of their medical colleges so that the instructors and students might go to the front. Later they saw the folly of such a course. Let us learn from their failures. This warning is especially applicable to the South, where the faculties are never so large as in the East.

England.[6]—English physicians have given themselves to the army so freely * * * that in some of the more populous districts there is but one physician for 6,000 people left in England.

Draft of Physicians.[7]—Claiming that the volunteer system of recruiting physicians for the Medical Reserve Corps of the Army has proven unsuccessful, Major Karl Connell and * * * have presented to the Council of National Defense a report urging the drafting of physicians selectively. * * *

Birmingham, Alabama.[8]—Dr. Frank Grace and Dr. Walter F. Scott, both of Birmingham, have been called into active service and ordered to the training camp at Fort Oglethorpe, Ga.

Dr. Seale Harris, of Birmingham, while attending the meeting of the American Medical Association in New York, became ill and was forced to undergo an operation for appendicitis.

Dr. J. S. McLester, of Birmingham, was operated on for appendicitis recently and is rapidly recovering.

1. Editorial: The Need for Medical Officers in the Army and Navy. Sou. Med. Jour., **10**:360 (April) 1917.
2. Editorial: The Physician's Opportunity for Service. Ibid., p. 443, May, 1917.
3. Editorial: The Conservation of America's Food Supply. Ibid., p. 445.
4. Ibid., p. 583, June.
5. Ibid., p. 533.
6. Ibid., p. 614, July.
7. Ibid., p. 616.
8. Ibid., p. 620, July.

Book Reviews

Time and the Physician. The autobiography of Dr. Lewellys F. Barker. New York: G. P. Putnam's Sons, 1942. Cloth $3.00.

Dr. Lewellys F. Barker, modest self-effacing autobiographer, in "Time and the Physician," was so careful to praise others that he forgot to mention many of his own achievements. He leaves it to the reader to learn from others that he was a worthy successor to Sir William Osler, not only as Professor of Medicine in Johns Hopkins Medical School, and Physician-in-Chief of Johns Hopkins Hospital, but that for three decades he influenced the progress of scientific medicine more than any other American medical teacher and practitioner in the Twentieth Century. He also failed to mention that he inspired the young medical men with whom he came in contact, and that he helped many of them financially

when they were hospital interns, and in the lean years when they were struggling to become established in the practice of medicine.

Though the most successful medical consultant in history, Dr. Barker did not forget, in his autobiography, to portray the price he paid for fame and fortune, when he wore patched trousers in the laboratories and hospital wards of Johns Hopkins Hospital. In relating the simple facts pertaining to his early education Dr. Barker reveals, what few have suspected, that, though he was the most carefully and the most thoroughly educated man of the second generation of Hopkins professors, he has suffered from an inferiority complex because he went into medicine from high school, via a druggist's apprenticeship, instead of from college. Certainly no one in reading Dr. Barker's books, or listening to his finished clinical bedside discussions, would suspect that he was anything less than a Ph.D. from a great university before he began the study of medicine. His brilliance and phenomenal memory are conspicuous traits.

If one were to criticize "Time and the Physician" one would say that the author devoted too much space to the history of the Johns Hopkins Medical School, which has been written and rewritten in the biographies of Osler, Welsh, Thayer, Halstead, Finney, Hugh Young, Abraham Flexner, and "Miss Lucy Slagle"; and that he gave too little space to the discussion of his own achievements. It also should be said that Dr. Barker's autobiography is the interesting and absorbing story of one of the chief actors in the dramatic development of modern medicine.

The Treatment of Burns. By Henry N. Harkins, M.S., M.D., Ph.D., F.A.C.S., Associate Surgeon, Henry Ford Hospital, Detroit. 457 pages, illustrated. Springfield, Illinois: Charles C. Thomas, Publisher, 1942. Cloth $6.50.

This book presents an exhaustive digest of knowledge derived from the experience and records of world authorities on burns. It has been carefully collected and assembled, and presents the latest information on new discoveries, including the latest innovations of treatment and recent developments in the care of burns obtained from the English during the present World War.

It is the most comprehensive work of its kind on both the theoretical and practical side, and is highly recommended as an invaluable adjunct to those interested in this subject.

Surgery of the Ambulatory Patient. By L. Kraeer Ferguson, A.B., M.D., F.A.C.S., Lieutenant-Commander, Medical Corps, United States Naval Reserve; Assistant Professor of Surgery, University of Pennsylvania. 923 pages, illustrated. Philadelphia: J. B. Lippincott Company. Cloth $10.00.

This work embraces a much wider scope in surgery than the average book on minor surgery. It is divided mainly into three parts. The first is a more or less general discussion of typical lesions of the ambulatory surgical case, with a description of their cause, course, and care. The second part deals with regional surgery, describing methods of surgical procedures along with the other treatment recommended. The third part considers fractures and dislocations, and their treatment in ambulatory cases.

There is an exceptionally good section on varicose veins and ulcers.

The book is well illustrated throughout, is written on thick glossy paper, and has an easily readable print.

For practical reference work to the young surgeon, its use is highly recommended.

Clinical Roentgenology of Pregnancy. By William Snow, M.D., Director of Radiology, Bronx Hospital, New York City. 171 pages, illustrated. Springfield, Illinois: Charles C. Thomas, Publisher, 1942.

This book is devoted to the discussion of the use of x-ray in pregnancy; position of the patient and technic of exposures; x-ray pelvimetry and cephalometry computations; the pelvic shape and its measurements; the fetus and pelvis; x-ray visualization of the soft tissues in pregnancy; and twenty-two case reports. The author has given more attention and more thought to the x-ray visualization of soft tissues in pregnancy than to any other portion of his book. Most of the 119 illustrations are very good. At this time, when it its important to conserve paper, the author might have placed his case reports beneath the illustrations, rather than on the opposite page. His method of x ray pelvimetry and cephalometry is interesting and valuable.

Electrocardiography. By Louis N. Katz, A.B., M.D., Director of Cardiovascular Research, Michael Reese Hospital, Chicago, Illinois; Assistant Professor of Physiology, University of Chicago, Chicago, Illinois. 580 pages, illustrated. Philadelphia: Lea & Febiger, 1941. Cloth $10.00.

The advances made during the past decade in the localization of myocardial injuries are well summarized. There are many excellent curves illustrating the different types of lesions resulting from coronary artery obstruction. There is also an adequate discussion of those conditions such as pulmonary embolism and pericarditis which are likely to be confused with coronary thrombosis.

The electrocardiographic aspects of cardiac arrhythmias are covered in detail. The value of the book would be considerably enhanced if there were a more comprehensive discussion of the clinical aspect of these irregularities.

The author recognizes clearly that the electrocardiographic method has its limitations and that its results need to be integrated with the clinical picture. He emphasizes a point which is often forgotten namely, that although the electrocardiogram is an objective phenomenon, the interpretation of it is subjective and hence entirely dependent upon the skill and experience of the interpreter. This point needs emphasis, for one frequently finds physicians who put entirely too much faith in the electrocardiographic method as such, and who fail to realize that the interpretation of the waves requires just as much skill as the interpretation of heart murmurs. This question is such an important one that it is well to quote Dr. Katz' point of view about it: "In short, the electrocardiograph is not a tool for the unscrupulous or a plaything for the erudite, nor is it an instrument of precision which replaces the ordinary clinical examination. It is a precise tool, an objective recording of events. However, the interpretation of the electrocardiograph is as subjective as any other

form of clinical interpretation, and its value depends just as much on the ability and experience of the cardiographer as the results of clinical examination depend on the ability and experience of the clinician. When its value and its limitations are thoroughly appreciated, and the two balanced, it is apparent that the electrocardiograph has a very definite place in clinical practice, a place that is becoming more and more clearly defined as our experience with it grows. It is as much of an error to expect too much from this tool as to ignore the valuable information that it can give."

If everyone who deals with electrocardiograms would saturate himself thoroughly with the philosophy ably presented here by Dr. Katz, one would see fewer patients whose lives had been made miserable by unnecessary restrictions imposed entirely on the basis of minor electrocardiographic changes.

Workmen's Compensation and the Physician. A Manual for the Use of General Practitioners and Insurance Carriers. By Henry H. Jordan, M.D., Associate Orthopedic Surgeon, Lenox Hill Hospital, New York, New York, with a Discussion of Traumatic Neuroses by Paul H. Hoch, M.D. 180 pages, illustrated. New York: Oxford University Press, 1941. Cloth $3.00.

This manual, which discusses the relationship of the practitioner to the workmen's compensation acts, shows how practitioners can cooperate with the insured and how to overcome the problems and difficulties that come up in this type of practice. The writer has a great deal of experience in this field. Stress is given to the first examination, treatment and the making of adequate notes. Roentgen examination is thoroughly discussed as is the evaluation of injury. Treatment is given ample consideration.

Communicable Disease Controls. A Volume for the Health Officer and Public Health Nurse. By Gaylord W. Anderson, A.B., M.D., Professor and Head of the Department of Preventive Medicine and Public Health, University of Minnesota, and Margaret G. Arnstein, R.N., M.A., M.P.H., District Supervising Nurse, New York State Department of Health. 434 pages, New York: The Macmillan Company, 1941. Cloth $4.25.

The history of communicable diseases and the teaching of methods of control that have been used in the past, the infective processes and methods of spread, control measures and the role of the law and the various agencies are discussed in this volume. The methods of epidemiologic investigation are outlined. There are descriptions of the individual diseases and the control of each, and of measures for community protection.

School Health Services. A Study of the Programs Developed by the Health Department in Six Tennessee Counties. By W. Frank Walker, Dr.P.H., and Carolina R. Randolph. New York: The Commonwealth Fund, 1941. Cloth $1.50.

This book answers the question as to how effective school services can be. Six counties in Tennessee were selected and studies were made of the various defects and the immunization status of the pupils. As a reward for acquiring a health rating, immunity states and health habits, the child was given a blue service bar and his school received suitable recognition. One of the difficulties is that the burden of making the examination is great. But a contribution is made by them in communicable disease control and over 50 per cent of the children will have defects corrected in order to get a blue ribbon. This is an important contribution to child health work.

Behind the Mask of Medicine. By Miles Atkinson. 347 pages. New York: Charles Scribner's Sons, 1941. Cloth $3.00.

Dr. Atkinson tells in the first chapters the why and wherefore of medicine, tracing its course from the earliest origins. He tells of the amazing progress that has been made in this past century and gives his opinion that medicine is suffering from a large amount of progress, that on this overdose medical practice is floundering about trying to get its bearings and take what is good to be incorporated into the accepted practice. He tells how the physician arrives at a diagnosis, his care of the patient to the exclusion of all else, even to the exclusion of euthanasia. Treatment, including some of the more blatant quackeries, are given. Surgery with its manifold operations and excesses in operations, but at the time its virtues are laid open to inspection. Specialism takes a "rap" for its overgrowth. Methods of practice, hospitals, socialized medicine and the future are the titles of other chapters. Neither physician nor layman will agree with all the author has written, but either will be stimulated to new thoughts and mental processes.

Who is My Patient? A Religious Manual for Nurses. By Russell L. Dicks, B.D. 149 pages. New York: The Macmillan Company. Cloth $1.50.

The author estimates that only about 20 per cent of the spiritual needs of the sick are cared for by the clergy. That leaves 80 per cent unattended. What is to be done about them? The doctor carries a great part of this ministry, but it is the nurse, who, both in the hospital and in the home, has a much better opportunity to observe the patient's spiritual condition and to minister to it. Such a ministry will not only give comfort to the patient but will also have a definite therapeutic value and may eventually result in material as well as spiritual benefit. The author points out how the good nurse will do not only what is assigned her from a sense of duty but will also be alert to detect spiritual difficulties on the part of patients. He makes definite suggestions as to how the nurse may minister to the patient through the art of listening and giving reassurance, by reading appropriate selections from the Bible, by sometimes praying with and for him, by securing the services of a minister, priest or rabbi and by preparing the patient and the sick room for his visit. As the sub-title of the book says, it is "A Religious Manual for Nurses," and, to my mind, it has a place not only in the curriculum of student nurses, but also in the library of the physician. Its main value for both should be in the direction of creating a better appreciation of the religious needs of sick people, which for the most part are totally neglected. Russell Dicks dedicates his book "To Dottie, my wife, who, as a nurse, knew the value of these things." Dicks, himself, was Chaplain for eight years of the Massachusetts General Hospital and was co-author with Dr. Richard Cabot, of "The Art of Ministering to the Sick." His little book is a modest, but significant, contribution to this field and deserves consideration.

Diarrhea in Infancy

Take It In Time

Just a day or two of light nourishment prepared from Mellin's Food as suggested below will usually avert an intestinal disturbance that might develop into a serious diarrhea if not taken in hand at the first appearance of loose stools.

Mellin's Food*. . 4 level tablespoonfuls
Water (boiled, then cooled) . . . 16 ounces

Give one to three ounces every hour or two until the stools lessen in number and improve in character.

The mixture may then be strengthened by the gradual substitution of boiled skimmed milk for water until the quantity of skimmed milk is equal to the normal quantity of milk used in the baby's formula. Finally the fat of the milk may be gradually replaced by skimming less and less cream from the milk.

Samples sent to physicians upon request.

Directions for using Mellin's Food are left entirely to the physician.

Mellin's Food Company, Boston, Mass.

**MELLIN'S FOOD: Produced by an infusion of Wheat Flour, Wheat Bran and Malted Barley admixed with Potassium Bicarbonate — consisting essentially of Maltose, Dextrins, Proteins and Mineral Salts.*

Southern Medical News

ALABAMA

Dr. Harvey B. Searcy, Tuscaloosa, has been appointed a member of the Council of the Southern Medical Association from Alabama for a regular Council term of five years beginning in mid-November, the appointment having been announced recently by the President-Elect, Dr. Harvey F. Garrison, Jackson, Mississippi. Dr. Searcy succeeds Dr. E. W. Rucker, Jr., Birmingham, whose term will expire with the close of the Richmond meeting in November, and having served the constitutional limit is not eligible for reappointment.

Dr. Cabot Lull, Birmingham, was elected to the Board of Directors of the National Tuberculosis Association at its annual meeting held recently in Philadelphia, Pennsylvania.

Dr. Lloyd Noland, Fairfield, member of the State Committee of Public Health, and Dr. A. A. Walker, Birmingham, represented the Alabama State Medical Association in the House of Delegates at the recent annual meeting of the American Medical Association.

Dr. Marion T. Davidson, Birmingham, was elected President of the Howard College Alumni Association at a luncheon held in connection with the Alumni and Class Day during the commencement exercises.

Dr. John C. McLeod, Jr., and Miss Helen Jones, Auburn, were married April 2.

Deaths

Dr. A. R. Davis, Moundville, aged 85, died recently.

Dr. Morriss DeWitt Kelley, Birmingham, aged 64, died recently of injuries received in a fall.

Dr. Jeremiah M. Lindsey, Ranburne, aged 72, died recently.

Dr. James Ernest Walker, Huntsville, aged 58, died recently.

Dr. L. A. Weaver, Rogersville, aged 82, died recently.

ARKANSAS

Arkansas Medical Society at its recent annual meeting installed Dr. R. B. Robins, Camden, President; and elected Dr. S. J. Allbright, Searcy, President-Elect; Dr. L. T. Martin, Hot Springs National Park, First Vice-President; Dr. R. C. Dickinson, Horatio, Second Vice-President; Dr. S. A. Drennen, Stuttgart, Third Vice-President; Dr. Paul L. Mahoney, Little Rock, Treasurer; and Dr. W. R. Brooksher, Fort Smith, Secretary.

Arkansas Tuberculosis Association has elected Dr. A. C. Shipp, Little Rock, President; Dr. J. D. Riley, State Sanatorium, Vice-President; Dr. Jerome S. Levy, Little Rock, Secretary; Dr. Howard Dishongh, Little Rock, Executive Committeeman; and Dr. R. M. Eubanks and Dr. H. A. Higgins, both of Little Rock, Directors.

Fourth Councilor District Medical Society has elected Dr. Marion Leverett, McGehee, President; and Dr. W. A. Snodgrass, Jr., Pine Bluff, Secretary.

Dr. A. C. Kolb, Little Rock, has been appointed Arkansas member of the Committee on Public Education of the American Psychiatric Association.

Dr. R. L. Taylor has been elected a Director of the Conway Chamber of Commerce.

Dr. Guy Hodges has been elected Surgeon of the Rogers Post of the American Legion.

Dr. J. Harry Hayes, Little Rock, has been elected Governor of District 7-B, Lions Clubs of Arkansas.

Dr. E. J. Byrd has moved from Bearden to Camden.

Dr. S. R. Livingston has moved from DeQueen to Santa Rita, New Mexico.

Continued on page 38

THE NEW YORK POLYCLINIC
MEDICAL SCHOOL AND HOSPITAL
(ORGANIZED 1881)

(The Pioneer Post-Graduate Medical Institution in America)

UROLOGY

A combined full time course in Urology, covering an academic year (8 months). It comprises instruction in pharmacology; physiology; embryology; biochemistry; bacteriology and pathology; practical work in surgical anatomy and urological operative procedures on the cadaver; regional and general anesthesia (cadaver); office gynecology; proctological diagnosis; the use of the ophthalmoscope; physical diagnosis; roentgenological interpretation; electrocardiographic interpretation; dermatology and syphilology; neurology; physical therapy; continuous instruction in cysto-endoscopic diagnosis and operative instrumental manipulation; operative surgical clinics; demonstrations in the operative instrumental management of bladder tumors and other vesical lesions as well as endoscopic prostatic resection.

FOR THE

General Practitioner

Intensive full time instruction in those subjects which are of particular interest to the physician in general practice. The course covers all branches of Medicine and Surgery.

FOR INFORMATION ADDRESS

MEDICAL EXECUTIVE OFFICER, 345 West 50th Street, NEW YORK CITY

GREEN AND RED LABEL DRIED BREWERS' YEAST

After all done, all written, grain grown dried brewers' yeast remains the most potent, concentrated source not only of the whole vitamin B complex but the whole of the pellagra preventive factor. Although nicotinic acid and riboflavin are important parts of the vitamin B complex and the pellagra preventive factors they are, alone, inadequate parts.

Following the discovery of the causes, relief and prevention of pellagra by Goldberger, Wheeler and their associates, Federal, State and County health officials and neighborhood physicians got busy. There is no reason why pellagra, within the next few years, cannot be ended and prevented.

Entering the campaign in 1927 with a supply of dried brewers' yeast potent in Goldberger's P-P (pellagra-preventive) factor and the full vitamin B complex, we wrote and widely distributed the following:

> "Pellagra is caused by a one-sided diet. Let all note this. Let no one urge one single product to the exclusion of everything Dr. Goldberger and his associates found valuable. Human life is too important to not be informed of every possible prevention and relief. Those afflicted with pellagra, or eating a diet which causes it, need more milk and eggs, more lean meat and dried grain yeast, vegetables and fruits."

Again we wrote:

> "Conservatism must be the rule in projecting products for use in human disease. The recovery and life of the patient, the professional reputation of the physicians are at stake."

THE FIXED POLICY

The president of this company, for a long time in pure food and public health work and knowing that all disease must be accurately diagnosed and all needed treatment had, has insisted that the use of dried brewers' yeast and other vitamin products in disease shall be restricted to medicine.

It is from such foundation that Vitafood Dried Brewers' Yeast continues to be presented to the physicians for use in pellagra and for use to furnish the whole of the dried brewers' yeast B complex.

There are two types of Vitafood Dried Brewers' Yeast. The Red Label has had the hop flavor substantially removed; whereas the Green Label retains the hop flavor.

Samples for clinical or professional use will be sent on request.

VITAMIN FOOD COMPANY, INC.
VITAMIN RESEARCH LABORATORIES, INC.

122 Hudson Street New York, N. Y.

Continued from page 36

Dr. Ulys Jackson, Harrison, has been transferred to Blytheville as Health Director for Mississippi County.

Dr. G. F. Hollingsworth has moved from Wildcat Mountain Sanatorium, Fort Smith, to Hampton.

Dr. D. V. Smith has moved from Huttig to Crossett.

Dr. Watten S. Riley, El Dorado, has been promoted to Major, Army Medical Corps, and is now serving overseas.

Dr. H. V. Kirby, Harrison, has been called to active duty as Lieutenant, Army Medical Corps, and assigned to Camp Shelby, Mississippi.

Dr. R. H. Johnson, Clarksville, now stationed in New Orleans, Louisiana, has been promoted to Major, Army Medical Corps.

Dr. Joe E. Beasley, Blytheville, has been called to active duty in the Naval Medical Corps as Lieutenant and has been assigned to the Washington Naval Base Hospital.

Dr. Guy Shrigley, Clarksville, has been appointed Lieutenant, Army Medical Corps, and assigned to Hoff General Hospital, Santa Barbara, California.

Dr. W. M. Woods, Huntington, has been called to duty as Lieutenant, Army Medical Corps, and assigned to the air force at Alexandria, Louisiana.

Dr. J. B. Elders, Walnut Ridge, Captain, Medical Corps, has been transferred to the 827th Tank Destroyer Battalion, Camp Forrest, Tennessee.

Dr. George F. Atkinson, Manila, has been called to active duty as 1st Lieutenant, Army Medical Corps, and assigned to the Presidio, San Francisco, California.

Dr. Guy P. Slaughter, Little Rock, has been ordered to active duty as Lieutenant, Army Medical Corps, and assigned to Letterman General Hospital, San Francisco, California.

Dr. Jim McKenzie, Hope, has been called to active duty as 1st Lieutenant, Army Medical Corps, and assigned to Bowman Field, Louisville, Kentucky.

Dr. T. K. Mahan, Blytheville, has been called to active duty as 1st Lieutenant, Army Medical Corps, and assigned to the Presidio, San Francisco, California.

Dr. Max Baldridge, Conway, has been called to active duty as Lieutenant, Naval Medical Corps, and assigned to Paris Island, South Carolina.

Dr. O. B. Barger, Mountain Home, has been called to active duty as Lieutenant, Army Medical Corps, and assigned to Fort Bliss, Texas.

Dr. B. V. Raley, Little Rock, has been called to duty as Lieutenant, Naval Medical Corps, and assigned to Naval Hospital, Pensacola, Florida.

Dr. Charles H. Finney, Captain, Army Medical Corps, formerly of Fort Smith, has been assigned to a station hospital for overseas duty.

Dr. A. G. Sullivan, Hot Springs National Park, has been called to active duty as Lieutenant-Commander, Naval Medical Corps, and assigned to Naval Hospital, Pensacola, Florida.

Dr. Euclid M. Smith, Hot Springs National Park, has been called to active duty at Ellington Field, Texas, as Major, Army Medical Corps.

Dr. Guy P. Slaughter and Dr. Pauline M. Kearney, both of Little Rock, were married recently.

Dr. Robert J. Haley, Sr., Paragould, and Mrs. Jewell Bradsher were married recently.

Deaths

Dr. Frank A. Gray, Batesville, aged 64, died May 18.

Chicago Eye, Ear, Nose & Throat College
Established 1897
231 W. Washington St., Chicago, Ill.
Practical postgraduate course in Ophthalmology and Otolaryngology.
Doctors admitted at any time for review and clinical observation.
OSCAR B. NUGENT, M.D., Director

Dr. Orlando Conrad Hankinson, Pine Bluff, aged 75, died April 14.

Dr. William Robert Haynie, West Memphis, aged 79, died recently.

Dr. Edgar E. Hodges, Little Rock, aged 62, died recently.

Dr. James William John, Pine Bluff, aged 74, died May 7.

Dr. Samuel Arthur Looper, Hot Springs National Park, aged 67, died recently.

Dr. Albert H. Miers, Mena, aged 51, died recently of coronary thrombosis and edema of the lungs.

Dr. Orlie Parker, Wabash, aged 60, died May 11 of cerebral hemorrhage.

Dr. Ernest Harl White, Little Rock, aged 54, died April 11.

DISTRICT OF COLUMBIA

Georgetown Clinical Society has elected Dr. A. J. Connolly, President; Dr. Edward O'Brien, Vice-President; and Dr. R. C. Kirchner, Secretary-Treasurer, all of Washington.

Washington Academy of Surgery has elected Dr. Frederick C. Fishback, President; Dr. Fred R. Sanderson, Vice-President; and Dr. James W. Watts, Secretary-Treasurer, all of Washington.

Emergency Hospital, Washington, reelected Dr. Stanley D. Wills and Dr. A. B. Moore, President and Secretary, respectively; and elected Dr. John H. Lyons and Dr. William B. Marbury, members of the Board. Approximately $90,000 has been expended by the hospital for the care of indigent patients during the fiscal year. Officials of the hospital pledged the maintenance and service of 100 additional beds in the interest of civilian defense.

District Chapter of the National Foundation for Infantile Paralysis has reelected all officers for the coming year. Dr. Edward Larkin and Dr. George C. Ruhland, members of the Executive Committee; Dr. H. Lynn Colvin, Dr. Custis Lee Hall, Dr. Guy W. Leadbetter and Dr. Julius S. Neviaser, members of the Board, all of Washington.

Dr. T. J. Abernethy has returned to Washington from Station Hospital, Camp Claiborne, Louisiana, where he served from January to May as a member of the Pneumonia Commission of the Board for Investigation of Epidemic Diseases in the U. S. Army.

Dr. James W. Watts, Associate Clinical Professor of Neurosurgery at George Washington University School of Medicine, Washington, was awarded the John Horsley Memorial Prize of $600 by the University of Virginia School of Medicine recently for his paper on "Psychosurgery" written in collaboration with Dr. Walter Freeman, Washington.

Dr. George C. Ruhland, Washington, has been selected by the Community Chest Executive Council to serve as one of the general advisers during the ensuing year for the Washington Council of Social Agencies.

Dr. William H. Sebrell, Jr., Washington, was elected Treasurer of the American Institute of Nutrition at its recent meeting held in Boston.

Dr. Charles S. Bunker, who is in command of the Navy Medical School, Washington, has been promoted to Rear Admiral.

Deaths

Dr. Jefferson Davis Bradfield, Washington, aged 74, died recently.

Dr. James Beaty Griffith, Washington, aged 72, died May 6.

Dr. Charles Maddox Hammett, Washington, aged 71, died recently.

Dr. Ralph Ellis Murrell, Washington, aged 50, died recently of cerebral hemorrhage.

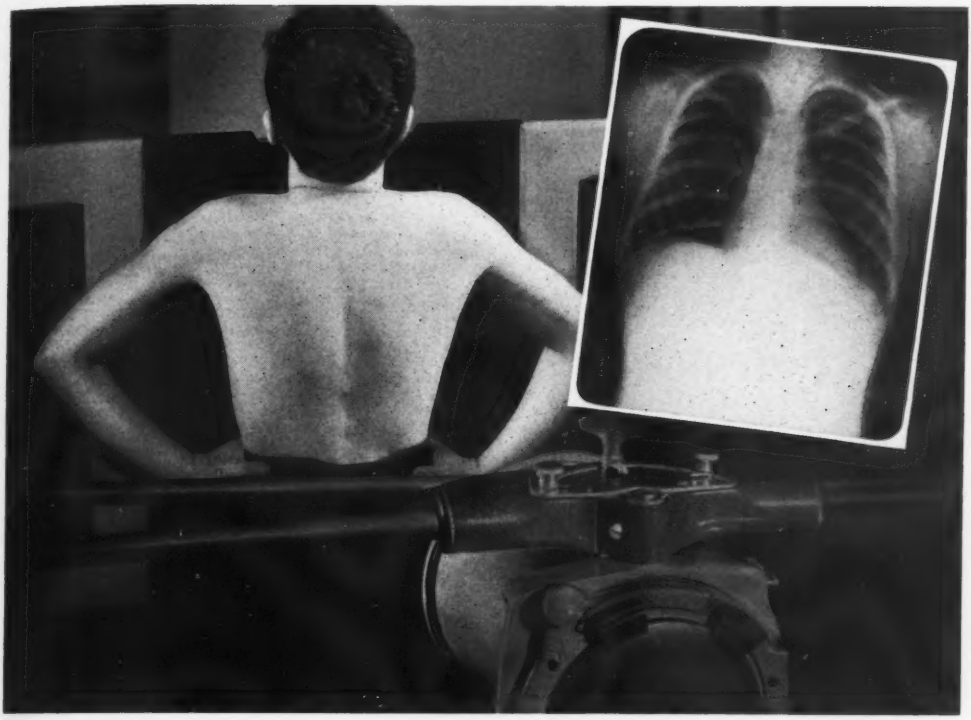
Dr. Nathan Norman Smiler, Washington, aged 53, died May 5.

FLORIDA

Dr. H. Marshall Taylor, Jacksonville, was elected President-Elect of the American Laryngological, Rhinological and Otological Society at the annual meeting in Atlantic City in June.

Florida Medical Association at its recent annual meeting installed Dr. Gilbert S. Osincup, Orlando, President; and elected

Continued on page 40



The diagnostic value of any radiograph is, in the final analysis, dependent upon the specialized knowledge of the one who makes the interpretation. This is the reason why you should refer your patients to a competent radiologist.

It is our belief that roentgenologic examination of the heart in cases of suspected coronary disease is an important adjunct to the other diagnostic methods which are generally employed. Moreover, it is our opinion that an accuracy of 75.7 per cent as judged electrocardiographically does not indicate the true value of roentgenologic examination.

** * * It not only affords confirmatory evidence, but furnishes additional objective information pertaining to associated cardiac lesions and pulmonary complications. It is, moreover, useful in detecting coronary disease when the electrocardiographic changes have not yet developed or have already disappeared.* — AM. HEART J., *16:* 141, 142, August, 1938.

EASTMAN KODAK COMPANY, Rochester, N. Y.

World's largest manufacturer of radiographic and photographic materials

Continued from page 38

Dr. Eugene G. Peek, Ocala, President-Elect; Dr. L. W. Blake, Bradenton, First Vice-President; Dr. Lloyd J. Netto, West Palm Beach, Second Vice-President; Dr. H. A. Walker, Miami Beach, Third Vice-President; and Dr. Shaler Richardson, Jacksonville, Secretary-Treasurer, reelected.

Florida Section of the American College of Surgeons met recently in Jacksonville. Dr. Frederick J. Waas, Jacksonville, is Chairman of the Florida State Executive Committee; Dr. Edward Jelks, Jacksonville, Secretary; and Dr. Walter C. Jones, Miami, Councilor.

Dr. Wilson T. Sowder, Tampa, formerly Health Officer of Hillsborough County, has been placed in charge of the Bureau of Venereal Disease Control of the State Board of Health. He succeeds Dr. Leo C. Gonzalez, Jacksonville, who has been in charge of the work for the last four years.

Dr. Edward Jelks, Jacksonville, Chairman of the Procurement and Assignment Committee, recently attended a conference of the Committee in Washington.

Dr. W. W. Wilson, Tampa, recently returned from a year's study at the Columbia University Skin Clinic.

Dr. Wade Martin Cline, Lakeland, and Miss Anita Hart Davies, Birmingham, Alabama, were married recently.

Dr. Ferdinand H. Kauders, Miami, and Miss Phyllis Salter were married recently.

Dr. Arthur E. Genius and Mrs. Charlotte Satterlee, both of Winter Park, were married recently.

Dr. John Phillips Moore, Ocala, and Miss Martha Helen Olsen were married recently.

Deaths

Dr. Paul Burmaster, Orlando, aged 74, died recently of coronary sclerosis.

Dr. Philip Finkle, Miami Beach, aged 47, died recently of coronary thrombosis.

Dr. Henry M. Meyer, Jupiter, aged 74, died recently of heart disease.

Dr. Earl C. MacCordy, St. Petersburg, aged 51, died recently.

GEORGIA

Dr. James E. Paullin, Atlanta, was elected President-Elect of the American Medical Association at the annual meeting in Atlantic City early in June.

Medical Association of Georgia at its recent annual meeting installed Dr. James A. Redfearn, Albany, President; and elected Dr. William A. Selman, Atlanta, President-Elect; Dr. Samuel J. Lewis, Augusta, and Dr. Cleveland Thompson, Metter, Vice-Presidents; Dr. Edgar D. Shanks, Atlanta, Secretary, reelected; and Dr. Allen H. Bunce, Atlanta, and Dr. Wm. A. Mulherin, Augusta, Delegates to the American Medical Association. The next meeting will be held in Atlanta.

Appling-Altamaha County Medical Society has elected Dr. E. J. Overstreet, President; Dr. H. C. McCracken, Vice-President; and Dr. J. T. Holt, Secretary-Treasurer, all of Baxley.

Stephens County Medical Society has elected Dr. S. E. Ayers, President; Dr. F. B. Welton, Vice-President; and Dr. C. L. Ayers, Secretary-Treasurer, all of Toccoa.

Georgia State Department of Health recently created an industrial hygiene service in its Division of Preventable Diseases with Dr. Lester M. Petrie, Atlanta, as Director.

Dr. T. F. Abercrombie, Atlanta, Director of Georgia Department of Public Health, was awarded the Hardman Loving Cup for his meritorious work to advance public health in the state.

Savannah dedicated its new Health Center building recently. It is a three-story building, constructed at a cost of $100,000 as a joint City-WPA project and houses all of the official and nonofficial or voluntary health agencies incorporated into the organization known as the Savannah Health Center. The President of the Center, Dr. A. J. Waring, has appointed the following to serve on various committees: Dr. Chas. C. Hedges, Dr. C. F. Holton, Dr. E. N. Maner, Dr. H. F. Sharpley, Jr., Dr. R. V. Martin, Dr. J. L. Elliott and Dr. Lawrence Lee, all of Savannah.

Emory University School of Medicine, Atlanta, has appointed Dr. Eugene A. Stead, Jr., formerly Instructor in Medicine, Harvard Medical School, Boston, as head of the Department of Internal Medicine.

Continued on page 42

PLASTIGUT

A New Universal Suture

truly non-reactive, non-capillary, guaranteed physiologically inert

An approach toward the ideal suture material is found in Plastigut Surgical Sutures, clinically developed by Dr. Joseph E. Bellas of Peoria. Plastigut is composed of synthetic plastic materials especially chosen with regard to suture requirements. Histological and clinical evidence proves it nonreactive, noncapillary and nonabsorbable. At any given stage, repair is more advanced in cases in which Plastigut is used than in those in which catgut is used. This is to be expected, since with Plastigut there is no irritating inflammatory and exudative reaction to hinder repair. There is no danger in leaving Plastigut in place; after two years it has been found intact, virtually a part of the supporting structures.

Plastigut is used in smaller sizes, due to its greater tensile strength. Plain Plastigut is offered in sizes No. 00, 0000 and 00000. Black, for skin work, is offered in sizes No. 0, 0000 and 00000. Size No. 00 is recommended for all general work, size No. 0000 for ties, size No. 00000 for plastic surgery and No. 0 for heavy tension sutures.

Price—Plastigut Sutures in Sterile Tubes, length 60 inches, all sizes. Dozen, $3.00. Gross, $29.50. Three-gross lots, per gross, $27.50.

References to the literature provided on request.

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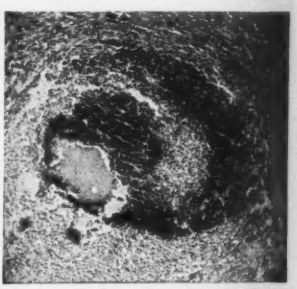
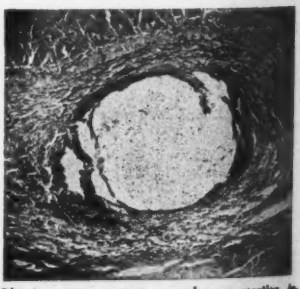
19th and Olive Sts. **ST. LOUIS, MISSOURI**



Plain catgut suture at two weeks; note absorption and inflammation.



Plastigut suture at two weeks; no reaction, inflammation or absorption.

AT ALL TIMES, IN ALL PLACES--

Your Patient Can Maintain His "Between-Meals" Schedule

High-tension war conditions and the strain of working under continued pressure seem to be causing flare-ups in peptic ulcer symptoms, spastic bowel and other conditions in which frequent feedings of bland, easily digested nourishment form an integral part of treatment.

The ambulant case, and particularly the patient who has to travel, will welcome the convenience of taking his "between-meals" milk ration in the form of

HORLICK'S
Malted Milk Tablets

Nourishing and Digestible

Each pleasant-tasting tablet yields 6 calories of balanced concentrated food essentials. A few dissolved in the mouth or chewed slowly help allay hunger and ward off ulcer pain.

Rich in Protective Factors

The tablets are supplied plain, chocolate flavored and fortified with Vitamins A, B_1, D, G.

And so at all times when the diagnosis suggests the need for bland, quickly digested nourishment at frequent intervals, remember Horlick's Malted Milk Tablets as well as

HORLICK'S

The *Complete* Malted Milk—Not Just a Malt Flavoring for Milk.

HORLICK'S

Continued from page 40

DEATHS

Dr. Alexander W. Burch, Lincolnton, aged 76, died recently.
Dr. Daniel Matthews Carter, Madison, aged 60, died recently.
Dr. William Edward Miller, Ludowici, aged 73, died recently.
Dr. Andrew Cornelius Dorminy, Hoboken, aged 61, died recently.
Dr. John Wade Chambliss, Americus, aged 61, died recently.
Dr. Edward Perrone Overby, East Point, aged 86, died recently.
Dr. John Chesley Stewart, Atlanta, aged 65, died recently.
Dr. Lester A. Rhyne, Atlanta, aged 74, died recently of chronic nephritis and uremia.

KENTUCKY

Dr. J. B. Lukins, Louisville, has been appointed a member of the Council of the Southern Medical Association from Kentucky for a regular Council term of five years beginning in mid-November, the appointment having been announced recently by the President-Elect, Dr. Harvey F. Garrison, Jackson, Mississippi. Dr. Lukins succeeds Dr. E. L. Henderson, Louisville, whose term will expire with the close of the Richmond meeting in November, and having served the constitutional limit is not eligible for reappointment.

Marion County Medical Society has elected Dr. Duncan Salot, Lebanon, President; Dr. S. F. Gregory, Bradfordsville, Vice-President; and Dr. N. D. Widmer, Lebanon, Secretary and Treasurer.

The Central State Hospital, built at Lakeland at a cost of $93,000, was recently opened.

Dr. Amplias W. Davis, Madisonville, has been appointed a member of the State Board of Health for a term ending December 31, 1947, succeeding Dr. Ernest L. Gates, Greenville.

Dr. Frank Marion Melton, Bradenburg, has been appointed Health Officer for the unit in Oldham County.

Dr. Woolfolk Barrow, III, of the Barrow Clinic, Lexington, has been called to active duty, Captain, Army Medical Corps, and is stationed at Egley Field, Florida.

Dr. Everett G. Grantham, Louisville, has reported at La Garde General Army Base Hospital, New Orleans, Louisiana, as attending neurosurgeon.

Dr. J. Duffy Hancock, Louisville, has been commissioned Major, U. S. Army, and will be stationed at Gorgas Hospital, Ancon, Canal Zone.

Dr. Fred W. Rulander, Louisville, has been commissioned in the Army Medical Corps and assigned to the New Orleans Army Base.

Dr. Charles K. Bush, Louisville, has been commissioned for service in the army and is stationed at Fort Hayes, Ohio.

Dr. John M. Keaney, Jr., Louisville, has been commissioned a 1st Lieutenant in the Army Medical Corps and is stationed at MacDill Field, Tampa, Florida.

Dr. J. C. Hill, Louisville, has been commissioned a Captain in the Medical Corps and is stationed at the 11th General Hospital, Camp Livingston, Louisiana.

Dr. E. C. Humphrey, Director of the Mercer County Health Department, recently reported for duty as Captain in the Army Medical Corps.

Dr. A. Clayton McCarty, Louisville, has reported to the Fifth Corps Area Headquarters, Fort Hayes, Columbus, Ohio, for special duty in the procurement and assignment of physicians and surgeons for the Medical Corps. He has been commissioned as Major.

Dr. R. Glen Spurling, Louisville, has reported to the Army Medical Center, Washington, to serve as neurosurgeon of the Walter Reed Hospital, and as Assistant Chief of Surgical Service.

Dr. Glenn Mitchell Shifley, Louisville, and Miss Geneva Peach, Pikeview, were married recently.

DEATHS

Dr. Joseph Ray Anderson, Louisville, aged 71, died recently.
Dr. Wilton F. Blackford, Louisville, aged 69, died recently of carcinomatosis with obstruction of the intestine.
Dr. Murison Dunn, Richmond, aged 70, died May 16.
Dr. Carl L. Prichard, Harold, aged 71, died recently of cerebral hemorrhage.

Continued on page 44

BILE ACID THERAPY

In Cholangitis

Acting directly upon the liver cells, Decholin (dehydrocholic acid) leads to an increase of 100 to 200 per cent in the amount of bile secreted. This hydrocholeretic action exerts a flushing influence upon the bile ducts,[1] and has been successfully employed in the treatment of noncalculous cholangitis for the removal of mucous and purulent accumulations.

Lauda[2] recommends an initial dose of Decholin sodium of 3 cc. (0.6 Gm.) for two days, together with antispasmodic treatment. If local symptoms of the bile ducts do not appear after the first injection, 10 cc. (2 Gm.) may be given on the third day, and repeated on the four following days. It is then advisable to interrupt this treatment for a short time. Oral treatment with Decholin tablets has a milder effect and is useful for prolonged medication.

Decholin and Decholin sodium are contraindicated in complete obstruction of the common or hepatic bile duct.

1. Ivy, A. C.: J. A. M. A. 117:1151 (Oct. 4) 1941.

2. Lauda, E.: Cholangitis, in Piersol, G. M.: Cyclopedia of Medicine, F. A. Davis Co., 1940 vol. 4 p. 228.

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Decholin

DISTRESSING AND POTENTIALLY DANGEROUS injuries such as minor burns require first-aid treatment for which 'S. T. 37' Antiseptic Solution is particularly well suited.

This oil-free, colorless, odorless preparation is not only highly bactericidal but exerts a mild surface analgesic effect as well when applied to raw, inflamed tissue. Moreover, 'S. T. 37' Antiseptic Solution is clinically non-toxic and its low surface tension facilitates penetration of minute recesses, thereby extending the field of its bactericidal action.

These characteristics make 'S. T. 37' Antiseptic Solution ideal for wet dressings in the treatment of burns. When 'S. T. 37' Antiseptic Solution is directly applied to such lesions its action is threefold:

1. **It exerts a potent bactericidal effect.**
2. **Pain is relieved by its surface analgesic action.**
3. **The tissues themselves are not further damaged and their normal physiologic activities are not adversely affected.**

'S. T. 37' Antiseptic Solution
is supplied in bottles of 5 and 12 ounces
SHARP & DOHME, Philadelphia, Pa.

The chemical composition of Karo in glass and in tins is identical

Non-Allergic Formulas

KARO is non-allergic even in corn-sensitive infants; it may be used safely in formulas prepared from evaporated, goat's or vegetable milk.

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CORN PRODUCTS REFINING CO.

17 Battery Place, New York, N. Y.

Continued from page 42

Dr. Joseph Harrison Riley, Lexington, aged 68, died recently of cerebral hemorrhage.

Dr. William Patrick Morse, Princeton, aged 69, died April 24.

Dr. Charles G. Russman, Louisville, aged 45, died April 29 of pneumonia.

LOUISIANA

Louisiana State Medical Society at its recent meeting installed Dr. Emmett L. Irwin, New Orleans, President; and elected Dr. C. C. deGravelles, New Iberia, President-Elect; Dr. John T. O'Ferrall, New Orleans, First Vice-President; Dr. W. V. Garnier, Bastrop, Second Vice-President; and Dr. E. H. Lawson, New Orleans, Third Vice-President; and Dr. P. T. Talbot, New Orleans, Secretary-Treasurer, reelected. Baton Rouge was selected for the next meeting place.

Louisiana Gynecological and Obstetrical Society has elected Dr. Earl Conway Smith, New Orleans, President; Dr. C. R. Mays, Shreveport, Vice-President; and Dr. Eugene Countiss, New Orleans, Secretary.

Louisiana Pediatric Society has elected Dr. S. George Wolfe, Shreveport, President; Dr. Chester A. Stewart, New Orleans, President-Elect; and Dr. W. C. Rivenbark, New Orleans, Secretary-Treasurer.

Louisiana Association of Pathologists has elected Dr. Maurice Couret, President; Dr. A. E. Casey, Vice-President; and Dr. Samuel H. Colvin, Secretary-Treasurer, all of New Orleans.

Louisiana Coroners' Association has elected Dr. Charles J. Barker, Thibodaux, President; Dr. Robert F. DeRouen, New Iberia, First Vice-President; Dr. M. M. Odom, Gretna, Second Vice-President; and Dr. L. L. Ricks, Independence, Secretary-Treasurer.

New Orleans Graduate Medical Assembly has elected Dr. H. Theodore Simon, President; Dr. Oscar W. Bethea, President-Elect; Dr. Waldemar R. Metz, First Vice-President; Dr. Donovan C. Browne, Second Vice-President; Dr. B. I. Burns, Third Vice-President; Dr. Joseph S. D'Antoni, Secretary; and Dr. Wm. H. Gillentine, Treasurer.

Dr. I. L. Robbins, Dr. Sidney Jacobs and Dr. Donovan C. Browne, New Orleans, were inducted as fellows of the American College of Physicians at its recent annual meeting.

Dr. Erasmus D. Fenner has completed fifty years of continuous active service on the visiting staff of the Charity Hospital, New Orleans.

Dr. George J. Taquino and Dr. C. Victor Vignes, New Orleans, had the honorary degree of Doctor of Science conferred on them by Loyola University at commencement exercises held in May.

Dr. F. J. Jaubert, New Orleans, has been reelected President of the Louisiana Society for Crippled Children.

Dr. Val Fuchs, New Orleans, was elected President of the Louisiana-Mississippi Ophthalmological and Otolaryngological Society at its recent meeting held in Jackson, Mississippi.

Dr. Ford S. Williams, U. S. Public Health Service, New Orleans, has been ordered for duty in San Francisco, California.

Dr. Carroll T. Bowen has been relieved from duty in Alexandria and assigned to the City Health Department, New Orleans.

Dr. Seth W. Kellam has been relieved from duty in New Orleans and ordered to Nashville, Tennessee.

Dr. W. H. Aufranc, Dr. W. P. Sellers, Dr. W. G. Budington, Dr. George G. Van Dyke and Dr. T. E. Morgan have been relieved from duty in New Orleans.

Deaths

Dr. Homer Chachere, Crowley, aged 83, died recently.

Dr. Henry Milburn Faust, Gold Dust, aged 68, died recently of gangrene of the left leg and arteriosclerosis.

Dr. Ford Nelson Jones, New Orleans, aged 59, died recently of heart disease.

Dr. Edward James Kevlin, Ponchatoula, aged 62, died recently of bulbar paralysis and amyothrophic lateral sclerosis.

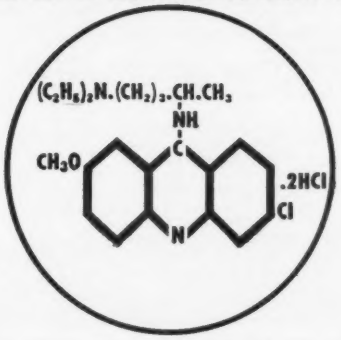
Dr. Fleater Palmer, Homer, aged 56, died recently of cerebral hemorrhage.

MARYLAND

Dr. William Houston Toulson, Baltimore, has been chosen Secretary of the Medical and Chirurgical Faculty of Maryland to fill the unexpired term of Dr. Richard T. Shackelford, Bal-

Continued on page 46

Synthesis IN OUR LABORATORIES



Acute attacks of malaria are checked rapidly — within a day or two.... Parasites in the blood stream disappear quickly — in a week or less. . . . The dose is small — in the average adult case only 1½ grains three times daily for five days.

Antimalarial Specific

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WINTHROP CHEMICAL COMPANY, INC.

Pharmaceuticals of merit for the physician

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Continued from page 44

timore, who has entered army service. Dr. Robert Lee Hall, Pocomoke City, is President. It was decided to cancel the semi-annual meeting of the Society which is usually held in the early fall.

Dr. Huntington Williams, Commissioner of Health of the City of Baltimore, who studied air raid medical administration in England for the Office of Civilian Defense, recently spoke on this subject at the annual postgraduate clinic at George Washington University School of Medicine, Washington, D. C.

Dr. George Finney, Colonel, Army Medical Corps, Baltimore, is Director of the 118th General Hospital Unit, one section of the Johns Hopkins University Hospital Unit, which the War Department recently activated and which will train at Camp Edwards, Massachusetts. Dr. Finney is the son of Dr. J. M. T. Finney, who headed this same numbered unit from Johns Hopkins when it went to France during the first World War.

Dr. Maurice C. Pincoffs, Colonel, Army Medical Corps, Baltimore, is Director of the 42d and 142d General Hospital Units from the University of Maryland Hospital which has been activated and will train at Fort Custer, Michigan, and Fort Riley, Kansas, respectively.

Dr. George W. Thorn, Associate Professor of Medicine, Johns Hopkins University School of Medicine, Baltimore, has been appointed the Hersey Professor of the Theory and Practice of Physic at Harvard Medical School, Boston, and Physician-in-Chief of the Peter Bent Brigham Hospital, succeeding the late Dr. Soma Weiss.

Dr. Thomas B. Turner, Professor of Bacteriology, Johns Hopkins University School of Hygiene and Public Health, Baltimore, and Lieutenant Colonel in the Army Medical Reserve Corps, has been called into active service as Chief of the Subdivision of Venereal Disease Control in the Surgeon General's Office.

Dr. Eli K. Marshall, Jr., Baltimore, was elected President of the American Society of Pharmacology and Experimental Therapeutics at its recent annual meeting.

Dr. Cyrus Eugene Hawks, Rockville, and Miss Eva Madora Hancock, Dodson, North Carolina, were married April 9.

Dr. Leonard J. Abramovitz, Baltimore, and Miss Jeanne Elizabeth Daubert, Tennersville, Pennsylvania, were married recently.

Dr. John Pennington Bond, Baltimore, and Miss Helen P. Book, Albemarle, were married recently.

Deaths

Dr. Edward Frost Giddings, Chevy Chase, aged 73, died recently.

Dr. John Gardiner Huck, Hydes, aged 50, died recently of cerebral hemorrhage.

Dr. Willey Clayton Nedrow, Friendsville, aged 58, died recently of uremia.

Dr. William Curtis Stifler, Baltimore, aged 69, died recently of cerebral hemorrhage.

Dr. Clifford Morrison Taylor, Westminster, aged 39, died recently.

Dr. Samuel Kendig Wallace, Baltimore, aged 40, died recently of massive hemorrhage

MISSISSIPPI

Dr. Ernest Jeff Holder and Miss Vivian Elizabeth Boylin, both of Prentiss, were married recently.

Deaths

Dr. W. S. Martin, Carthage, aged 70, died recently of chronic nephritis and valvular heart disease.

Dr. Eugie A. Ross, Dlo, aged 64, died recently of esophageal varices.

MISSOURI

Missouri State Medical Association at its recent annual meeting installed Dr. H. L. Kerr, Crane, President; and elected Dr. Andrew W. McAlester, Jr., Kansas City, President-Elect; Dr. R. M. James, Joplin, Dr. E. J. Schisler, St. Louis, and Dr. G. A. Lau, St. Joseph, Vice-Presidents; Dr. R. L. Thompson, St. Louis, Treasurer, reelected; and Mr. E. H. Bartelsmeyer, St. Louis, Executive Secretary, reelected.

Kansas City Association of Railway and Industrial Physicians and Surgeons have elected Dr. E. P. Heller, President; Dr. Carl H. Brust, Vice-President; Dr. H. L. Mantz, Secretary; and Dr. C. G. Vincent, Treasurer.

Missouri Chapter of the American College of Chest Physicians has elected Dr. Paul Murphy, Koch, President; Dr. Sam H. Snider, Kansas City, Vice-President; and Dr. J. A. Stocker, Mount Vernon, Secretary and Treasurer.

Henry County Medical Society has elected Dr. E. C. Peeler, President; and Dr. R. S. Hollingsworth, both of Clinton, Secretary and Treasurer.

Newton County Medical Society has elected Dr. Louis E. Rolens, Grandy, President; Dr. Roy C. Lamson, Neosho, Vice-President; and Dr. John A. Guthrie, Neosho, Secretary-Treasurer.

Dent County Medical Society has elected Dr. Lloyd H. Hunt, President; Dr. Wm. G. Dillon, Vice-President; and Dr. Fred E. Butler, Secretary, all of Salem.

St. Francois-Iron-Madison-Washington-Reynolds County Medical Society has elected Dr. J. L. Thurman, Potosi, President; Dr. J. P. Yeargain, Irondale, Vice-President; and Dr. C. C. Ault, Farmington, Secretary.

Contract has been let and work begun for the building of the New Health Center at the St. Louis County Hospital which will cost more than $500,000.

Dr. James C. Schumacher, St. Louis, has joined the U. S. Public Health Service for the duration of the war and is stationed in California.

Dr. Robert A. Ryan, Jr., Mountain Grove, and Miss Frances Isabel Sells, Ottumwa, Iowa, were married recently.

Dr. DeWitt Daniel Stuart and Miss Betty Sue Gardner, both of Brunswick, were married recently.

Continued on page 48

LaMOTTE BLOOD CHEMISTRY SERVICE

NEW and IMPROVED
LaMOTTE FALLING DROP DENSIOMETER
(Two Models)

An important adjunct in treating cases of shock.
For determining blood proteins, albumin - globulin ratio and specific gravity of body fluids. These units are produced exclusively with the cooperation of H. G. Barbour and W. F. Hamilton, and approved by them.
This outfit is now available in two models—the larger model comes complete for $65 and the small model at $35. Prices F.O.B. Towson, Md.

Send for complete information.

This service includes a series of similar outfits for conducting the following accurate tests: Blood Sugar, Blood Urea, Sulfathiazole, Sulfapyridine, Sulfanilamide, Sulfadiazine and Sulfaguanidine in Blood and Urine. Icterus Index, Phenolsulphonthalein, Urine pH, Blood pH, Gastric Acidity, Calcium - Phosphorus, Blood Bromides, Urinalysis.

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Metrazol

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INJECT 1 to 3 cc. Metrazol as a restorative in circulatory collapse, respiratory distress, deep anesthesia, and in morphine and barbiturate poisoning. In the emergencies of pneumonia and other over-whelming infections, and in congestive heart failure, give Metrazol, 1½ to 4½ grs., t. i. d.

Ampules 1 cc. and 3 cc. (each cc. containing 1½ grains Metrazol), Tablets 1½ grains and the soluble Powder

METRAZOL (brand of pentamethylentetrazol), Trade Mark and Pat. reg. U. S. Pat. Off.

Council Accepted

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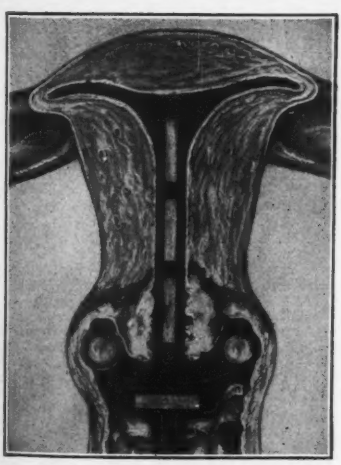
IMPROVE YOUR RESULTS IN CANCER OF THE CERVIX



CONSISTENTLY high percentages of 5-year cures in Carcinoma of the Cervix are reported by institutions employing the French technique illustrated here. Ametal rubber applicators encase the heavy primary screens and provide ideal secondary filtration to protect the vaginal mucosa. Radium or Radon applicators for the treatment of Carcinoma of the Cervix and provided with Ametal filtration are available exclusively through us. Inquire and order by mail, or preferably by telegraph or telephone reversing charges. Deliveries are made to your office or hospital for use at the hour you may specify.

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Continued from page 46

DEATHS

Dr. Edward Sherman Baker, Kansas City, aged 51, died recently of hypertensive heart disease.

Dr. Given Campbell, St. Louis, aged 74, died recently.

Dr. William Augustus K. Chase, St. Louis, aged 69, died recently of heart disease.

Dr. Joseph Gill, St. Louis, aged 78, died recently.

Dr. James W. Kinyoun, Independence, aged 83, died recently of arteriosclerotic heart disease and chronic nephritis.

Dr. James Jorgen Clausen, Kansas City, aged 76, died recently of carcinomatosis.

Dr. Oscar Clarence Kessler, Elkland, aged 82, died recently of nephritis.

Dr. Wilmer J. Sell, Crocker, aged 74, died recently of aortic insufficiency.

Dr. Herman August La Force, Joplin, aged 55, died recently.

Dr. John Elvin Parmer, Mokane, aged 72, died recently of chronic encephalitis.

NORTH CAROLINA

Dr. John Riley Black, Jr., and Miss Alma Jane Miller, both of Whitesville, were married recently.

Dr. James Pettigrew Bunn, Rocky Mount, and Miss Margaret Elizabeth Smith, Heathsville, were married recently.

Dr. James G. Lupo, Oteen, and Miss Meadie Johnston, Carrollton, Alabama, were married recently.

Dr. Robert Jennings Murphy, Jr., Goldsboro, and Miss Katherine Trigg Evans, Christiansburg, were married recently.

Dr John Luther Shirey, Mount Pleasant, and Miss Pegge Elaine Johnson, Birmingham, Alabama, were married recently.

The Tulane University of Louisiana

SCHOOL OF MEDICINE

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Combined preparation—One year externship, one year residency, no tuition charge. Men and women eligible.

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1430 Tulane Avenue, New Orleans, La.

Dr. Herbert Daniel Tyler, Charlotte, and Miss Jane Cook Coffin, Atlanta, Georgia, were married recently.

Dr. Joseph Thomas Wyche, Hallsboro, and Miss Elizabeth Calder, Charlotte, were married recently.

DEATHS

Dr. Lovelace B. Capehart, Raleigh, aged 78, died recently of heart disease.

Dr. Fred Lee Herbert, Andrews, aged 63, died recently of carcinoma of the pancreas.

Dr Charles Braxton Ingram, Mount Gilead, aged 84, died recently of cerebral hemorrhage.

Dr. Charles W. Moseley, Greensboro, aged 76, died recently of pneumonia.

Dr. John Jay Munden, Henderson, aged 77, died recently of coronary occlusion.

Dr. Seifert Clairmonte Pyle, Charlotte, aged 37, died recently of pneumonia.

Dr. Norman Grady Williams, Franklin, aged 49, died recently of cirrhosis of the liver.

OKLAHOMA

Oklahoma State Medical Association at its fiftieth annual meeting held recently in Tulsa installed Dr. J. D. Osborn, Frederick, President; and elected Dr. James Stevenson, Tulsa, President-Elect; Dr. Galvin L. Johnson, Pauls Valley, Vice-President: Dr L. J. Moorman, Oklahoma City, Secretary-Treasurer, reelected; and Mr. R. H. Graham, Oklahoma City, Executive Secretary, reelected. The next meeting will be held at Oklahoma City.

Stephens County Medical Society has elected Dr. A. J. Weeda, Duncan, President; Dr. E. H. Lindley, Duncan, Vice-President; and Dr. W. K. Walker, Marlow, Secretary-Treasurer.

Dr. Clifford C. Fulton, a Lieutenant Commander in the naval services, is stationed at the U. S. Naval Hospital, in Corpus Christi, Texas, where he was head of a Surgical ward before his promotion to head of the school. Dr. Fulton was formerly from Oklahoma City.

Dr. J. William Finch, Hobart, has been elected a Fellow of the American College of Physicians.

DEATHS

Dr. William Claude Sain, Ardmore, aged 57, died recently of tumor of the brain.

Dr. Thomas E. Shepard, Tulsa, aged 73, died recently of pneumonia.

SOUTH CAROLINA

South Carolina Medical Association at its recent annual meeting installed Dr. T. A. Pitts, Columbia, President; and elected Dr. W. A. Smith, Charleston, President-Elect; Dr. J. Warren White, Greenville, Vice-President; Dr. Julian P. Price, Florence, Secretary-Treasurer; and Dr. J. Howard Stokes, Assistant Secretary. The next annual meeting will be held at Spartanburg.

The Fourth Medical District has been divided, Spartanburg, Union and Cherokee Counties composing the new Ninth District.

Dr. J. H. Young, Columbia, recently completed a postgraduate course in eye, ear, nose and throat work at the Gills Memorial Clinic, Roanoke, Virginia.

Dr. Chas. Tripp, Easley, is Chairman of the State Highway Commission.

DEATHS

Dr. Harry McMahon Barton, Walhalla, aged 71, died recently.

Dr. Paul Eugene Payne, Columbia, aged 45, died May 4.

TENNESSEE

Dr. Kate Savage Zerfoss, Nashville, has been appointed a member of the Council of the Southern Medical Association from Tennessee for a regular Council term of five years beginning in mid-November, the appointment having been announced recently by the President-Elect, Dr. Harvey F. Garrison, Jackson, Mississippi. Dr. Zerfoss succeeds Dr. Horton Casparis, Nashville, whose term will expire with the close of the Richmond meeting

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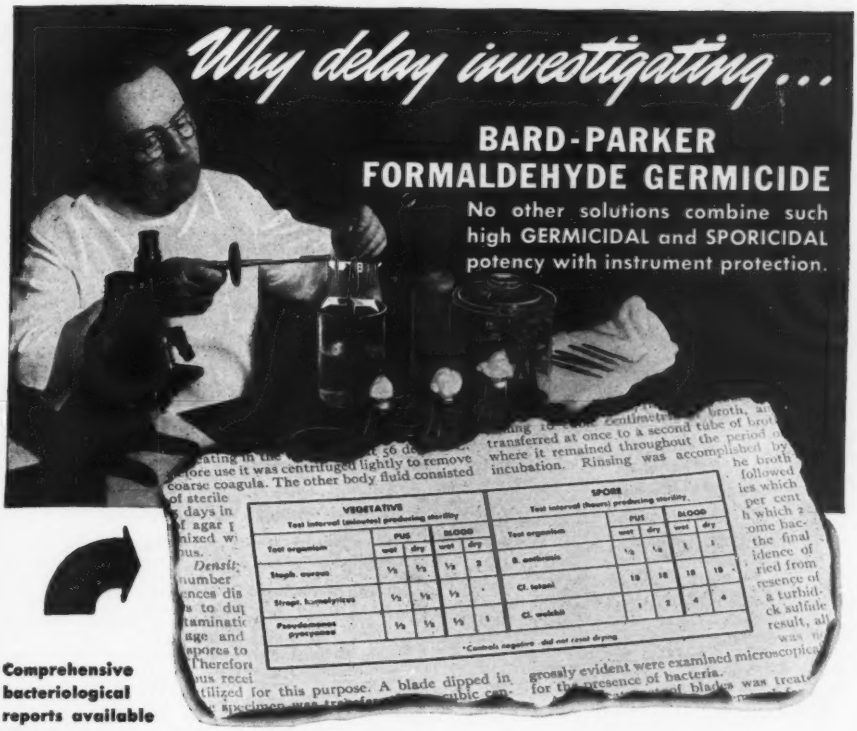

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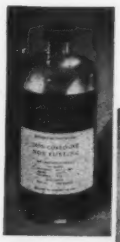

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Continued from page 48

in November, and having served the constitutional limit is not eligible for reappointment. Dr. Zerfoss is the daughter of the late Dr. Giles C. Savage, Nashville, one of the founders and an early president of the Southern Medical Association.

Meharry Medical College, Nashville, has been given a grant of $250,000 by the W. K. Kellogg Foundation, Battle Creek, Michigan, to finance an educational program in medicine, dentistry and nursing. Fifty thousand dollars will be made available annually for five years.

A bond issue of $500,000 has been provided, the proceeds of which are to be used for the construction of a tuberculosis hospital in Tennessee. The act providing the issue also authorizes the creation of a commission to make application for a grant of funds from the federal government to supplement the local funds and to select the location for the hospital.

Dr. Vernon W. Foster, formerly of Savannah, Georgia, has been placed in charge of the newly created health unit of Henry County.

Dr. Horace M. Roberson, formerly Health Director for Bledsoe and Sequatchie Counties, has been placed in charge of the health unit of Rhea and Meigs Counties, succeeding Dr. Hollis C. Miles, Dayton, who resigned to return to private practice in Cincinnati, Ohio.

Classified Advertisements

RATES for insertion in the Classified Column are as follows: $2.00 minimum, which includes the first 50 words; for each word in addition to the original 50 words, the charge is 3c.

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Dr. Loyal D. Farragut, Jackson, has resigned as Director of the Madison County Health Unit to enter army service.

Dr. James Garnett Bruce, Jr., Watts Bar Dam, and Miss Janet Floyd, Chattanooga, were married recently.

Deaths

Dr. Thomas H. Coke, Denver, aged 73, died recently of hypertension and hemiplegia.

Dr. Ada Barker Luttril, Knoxville, aged 61, died recently.

Dr. Bernice Turner Wright, Memphis, aged 39, died recently of an injury received in an automobile accident.

TEXAS

Dr. Eugene L. Aten, Dallas, was elected President of the Texas Society for Mental Hygiene at its recent annual meeting.

Deaths

Dr. George Monroe Cobb, Ector, aged 73, died recently.

Dr. James Arnold Bledsoe, Port Arthur, aged 53, died recently of heart disease.

Dr. Charles N. McGaffey, Dallas, aged 55, died recently of arteriosclerosis.

Dr. Henry Clay Perkins, Austin, aged 44, died recently of an automobile accident.

Dr. Josiah Houston Warnick, Abilene, aged 79, died recently of cerebral hemorrhage.

Dr. Claude N. Workman, Willowpoint, aged 67, died recently of hypertensive heart disease.

VIRGINIA

Hanover County Medical Society has elected Dr. J. A. Wright, Doswell, President; Dr. A. E. Murray, Beaverdam, Vice-President; and Dr. J. D. Hamner, Jr., Ashland, Secretary-Treasurer.

Roanoke Academy of Medicine has elected Dr. Paul Davis, President; Dr. A. M. Groseclose and Dr. Charles Irvin, Vice-Presidents; and Dr. Ruth Barnhart, Secretary-Treasurer, all of Roanoke.

South Piedmont Medical Society has elected Dr. Bernard H. Kyle, Lynchburg, President; Dr. James R. Gorman, Lynchburg, Dr. W. C. Brann, South Boston, and Dr. R. W. Upchurch, Danville, Vice-Presidents; and Dr. F. O. Plunkett, Lynchburg, Secretary-Treasurer.

Virginia Tuberculosis Association at its recent annual meeting elected Dr. Frank S. Johns, Richmond, President; and Dr. Roy K. Flannagan, Richmond, Secretary.

U. S. Army General Hospital Unit No. 45, formed at the Medical College of Virginia, Richmond, has been ordered to active duty. Dr. John Powell Williams and Dr. A. Stephens Graham, both of whom have been commissioned Lieutenant Colonels, are in charge of organizations of the unit, Lieutenant Colonel Williams being Commanding Officer of the Unit. During the first World War Base Hospital No. 45 was organized at the Medical College of Virginia under the leadership of Dr. Stuart McGuire of Richmond, and that unit performed distinguished service on the battlefields of France.

Kings' Daughters' Hospital, Portsmouth, has received a grant of $695,880 by the Federal Works Agency which will finance the addition of a four-story wing providing one hundred and thirty additional beds. The grant will also provide a new nurses' home and complete equipment.

Medical College of Virginia, Richmond, has appointed Dr. Jacques P. Gray, formerly Director of the Hillsdale County (Michigan) Department of Health, Hillsdale, Professor of Preventive and Public Health Medicine and Dean. Dr. Lee E. Sutton, Jr., who has been Dean at the Medical School since 1932, will continue as Professor of Pediatrics.

The Montgomery County School Board, Christiansburg, recently adopted resolutions requiring all children attending public schools next fall to present physicians' certificates stating that they have been examined by physicians and are free from chest illnesses. These certificates must be presented every three years thereafter. The group must also present certificates declaring that they are free from communicable diseases, these certificates having to be renewed every twelve months.

Members of the Virginia USO Sponsoring Committee recently appointed are Dr. Staige Blackford, University; Dr. Wyndham B. Blanton, Dr. Charles M. Caravati, Dr. Fred M. Hodges, Dr

Continued on page 52

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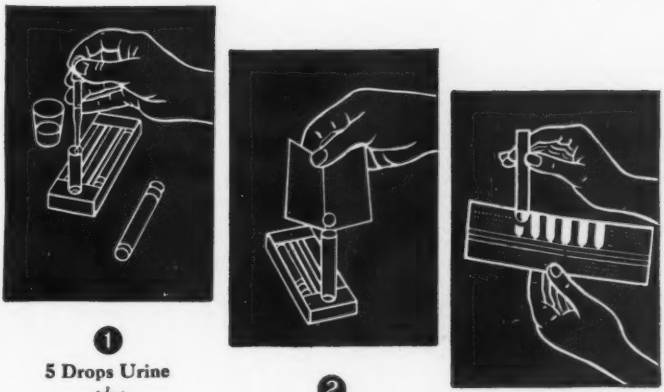

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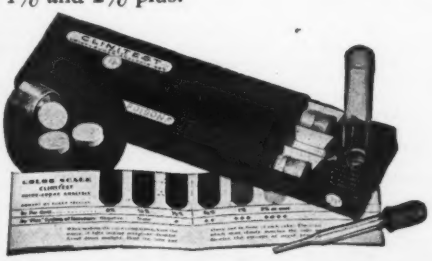

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Continued from page 50

J. Shelton Horsley, Dr. Stuart McGuire, Dr. Stuart N. Michaux, Dr. Clifton M. Miller, Dr. Roshier W. Miller, Dr. W. Lowndes Peple and Dr. Charles R. Robins, Sr., all of Richmond. The USO started a drive June 1 for $407,000 in Virginia to provide recreational and comfort facilities for men in the armed forces.

Dr. H. Hudnall Ware, Jr., Richmond, recently announced the association of Dr. W. C. Winn in the practice of obstetrics and gynecology with offices at 816 West Franklin Street. Dr. Winn for the past two years has been connected with the Bureau of Maternal and Child Health of the Virginia State Department of Health.

The following have been reappointed on the State Board of Medical Examiners for a term of four years: Dr. R. D. Bates, Newton, Dr. P. St. L. Moncure, Norfolk; Dr. H. U. Stephenson, Richmond; Dr. Wm. B. McIlwaine, Petersburg; Dr. I. C. Harrison, Danville; Dr. J. W. Preston, Roanoke; Dr. P. W. Boyd, Winchester; Dr. Lewis Holladay, Orange; and Dr. F. H. Smith, Abingdon.

The John Horsley Memorial Prize of $500 for the best thesis on a surgical subject has been awarded to Dr. James W. Watts, Associate Clinical Professor of Neurosurgery at George Washington University School of Medicine, Washington, D. C., who is a graduate of the University of Virginia School of Medicine, class of 1928. This award was established at the University of Virginia by Dr. J. Shelton Horsley, Richmond, as a memorial to his father and is bestowed upon a medical graduate every two years.

Dr. H. B. Mulholland, Charlottesville, has been appointed Councilor from the Eighth District by the President of the Medical Society of Virginia until the meeting in the fall to take the place of Dr. A. D. Hart, Charlottesville, who has entered the army service.

Dr. J. W. Preston, Roanoke, has been appointed Chairman of the Department of Clinical and Medical Education of the Medical Society of Virginia, succeeding Dr. Walter B. Martin, now in the army. Dr. Guy R. Fisher will act as Chairman of the Medical Economics Committee in place of Dr. John Hundley, Jr., also in the army.

Dr. Hunter H. McGuire, Winchester, has been reelected President of the Winchester Memorial Hospital, a position he has held for several years.

Dr. Edwin P. Lehman, Charlottesville, has been elected a member of the Board of Directors of the American Society for the Control of Cancer.

Dr. Eugene M. Landis, Charlottesville, was elected President of the American Society of Clinical Investigation at its recent meeting.

Dr. John O. Hurt, who has been Clinical Director at the Lynchburg State Colony, is now engaged in general practice in the Medical Arts Building, Roanoke.

Dr. George S. Stevenson, formerly Medical Director of the National Committee for Mental Hygiene, was recently elected President of the National Health Council, succeeding Dr. Kendall Emerson, Managing Director of the National Tuberculosis Association.

Dr. B. B. Bagby, formerly with the State Health Department and recently Chief Health Officer and Epidemiologist of Richmond, has been named Defense Director of Northumberland County pending the appointment of a permanent officer. Dr. Bagby will have complete charge of civilian defense activities in that county.

Dr. Francis J. Clements, Stony Creek, is Health Officer of Dinwiddie County which has joined with Sussex-Prince George Counties as a health district.

Dr. Daniel Hope, Jr., Lawrenceville, formerly Assistant Health Officer of the Brunswick-Greenville-Mecklenburg Health District, has been appointed Health Officer of the Alleghany-Botetourt Health District, with offices in Covington, succeeding Dr. Wyatt E. Roye, Covington, who has been transferred to the tuberculosis outpatient service of the State Health Department at Richmond.

Dr. Wallace E. Baker, Petersburg, has been appointed Assistant Epidemiologist in the Division of Venereal Disease Control of the State Department of Health. Dr. William E. Chapin, Richmond, succeeds Dr. Baker in his former position of Field Epidemiologist for the Camp Lee area with headquarters in Petersburg.

Dr. W. O. Bailey, Jr., Leesburg, has been promoted to Captain in the Army Medical Corps and was last reported stationed at Trinidad, British West Indies.

Dr. Dallas G. Sutton, who at present is in command of the Norfolk Naval Hospital, Portsmouth, has been promoted to Rear Admiral, Medical Corps, U. S. Navy.

Dr. James Oten Burke, Captain, Medical Corps, U. S. Army, and Miss Alice DeLancey Davis, Atlanta, Georgia, were married May 2.

Dr. Henry St. George Tucker, Jr., Richmond, and Miss Mary Grasty Bell, Staunton, were married recently.

Deaths

Dr. Frank Eubank Booker, Halifax, aged 67, died recently of cerebral hemorrhage.

Dr. James Noah Greear, St. Paul, aged 83, died recently.

Dr. William Binford King, Arlington, aged 54, died recently.

WEST VIRGINIA

Dr. Ray M. Bobbitt, Huntington, has been appointed a member of the Council of the Southern Medical Association from West Virginia for a regular Council term of five years beginning in mid-November, the appointment having been announced recently by the President-Elect, Dr. Harvey F. Garrison, Jackson, Mississippi. Dr. Bobbitt succeeds Dr. R. J. Wilkinson, Huntington, whose term will expire with the close of the Richmond meeting in November, and having served the constitutional limit is not eligible for reappointment.

Dr. Walter E. Vest, Huntington, was elected President of the American Therapeutic Society at its forty-third annual meeting held in Atlantic City early in June.

West Virginia State Medical Association will hold its annual meeting at the Prichard Hotel, Huntington, July 13-15. Mr. Paul V. McNutt, Federal Security Administrator, and Dr. Allan Roy Dafoe of Canada, will be the guest speakers.

The following physicians compose the Naval Medical Specialist Unit No. 40 at the U. S. Naval Hospital, Quantico, Virginia: Commander Wm. Marco Sheppe, Wheeling; Lieutenant Commanders H. G. Little, A. L. Oserman, J. K. Stewart, E. L. Armbrecht and A. L. Jones, all of Wheeling; Lieutenant Commander Russel Kessel, Charleston; Lieutenant Commander Ralph Boice, Parkersburg; and Lieutenant Commander Morris Reingold, Lynn, Massachusetts.

Dr. Albert H. Hoge, Bluefield, was reelected to the Board of Governors representing the State of West Virginia at the American College of Physicians' recent meeting.

Dr. R. C. Edson, formerly Assistant Superintendent and Co-Medical Director of Hopemont Sanitarium, has accepted a position as Assistant Director of Tuberculosis Control for the State of Connecticut and is now located at West Hartford, Connecticut.

Deaths

Dr. Harry Cockerille Blair, Elkins, aged 56, died recently.

Dr. Daniel H. Crawford, Charleston, aged 76, died recently.

Dr. Leonidas W. Humphreys, Huntington, aged 74, died recently.

Dr. Glenn Moomau, Petersburg, aged 61, died recently of coronary occlusion.

Dr. Harry N. Moser, Terra Alta, aged 69, died recently.

Dr. George Philip Naum, Glasgow, aged 40, died recently.

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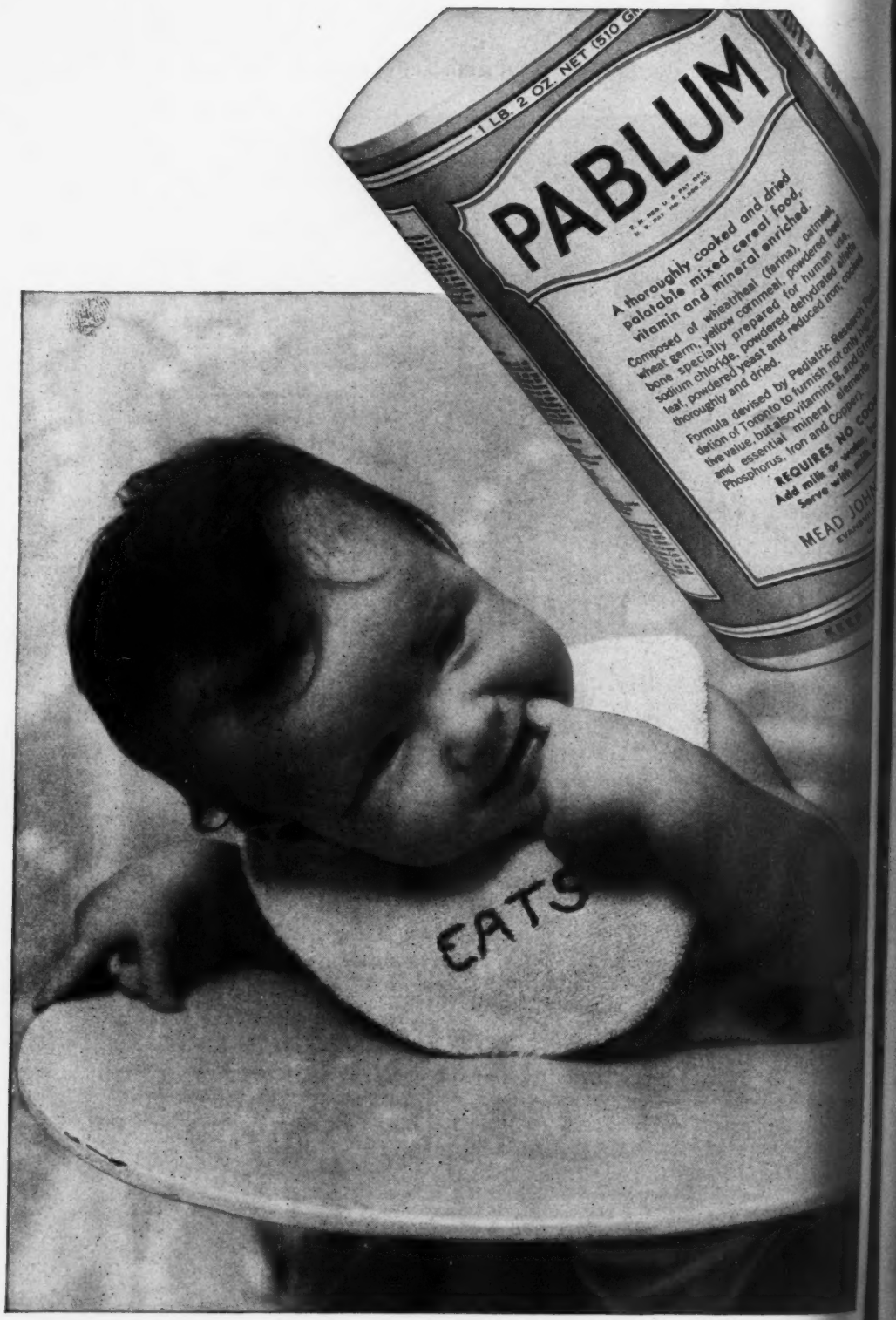
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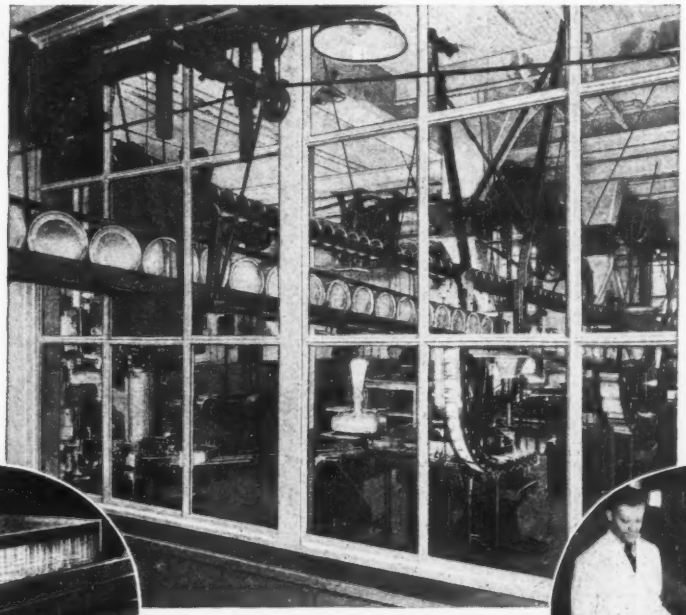

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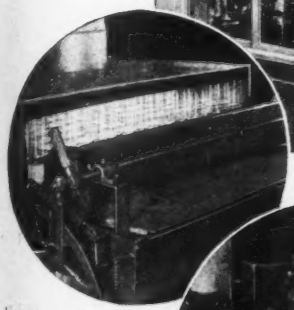

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